BULLETIN No. 142

Sugar Beet Diseases

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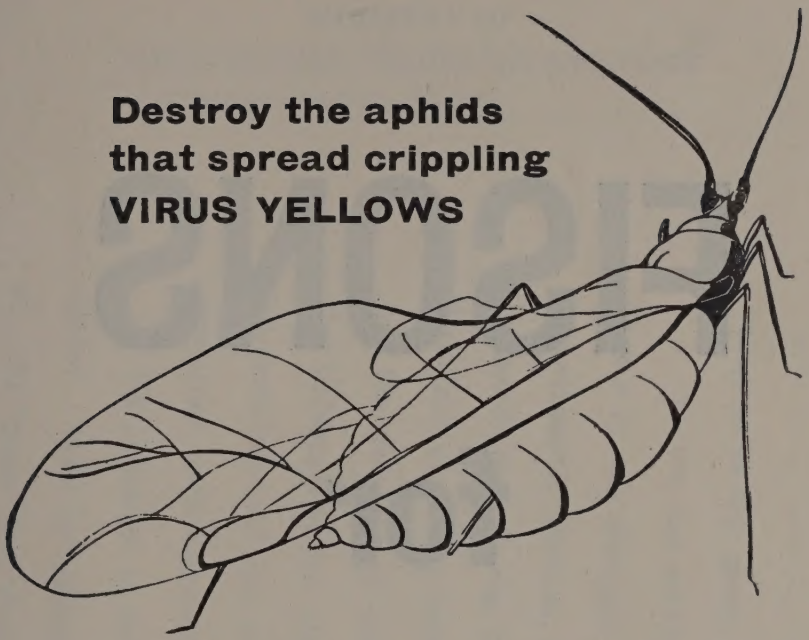

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MINISTRY OF
AGRICULTURE, FISHERIES AND FOOD

Sugar Beet Diseases

Bulletin No. 142

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Foreword

SUGAR beet cultivation is relatively new to Britain. Before 1912, when the first factory for sugar extraction was opened at Cantley, Norfolk, less than 500 acres were devoted to the crop, and it was not until after the government subsidy on home-produced sugar was introduced in 1924 that the acreage rose very rapidly within six years to 350,000. Diseases and pests soon made their presence felt, as they usually do when a new crop finds favour, and by the early thirties a number of them were already beginning to cause concern. Little was known about them, and when the Ministry issued its first Bulletin on the subject in 1935 most of the information it contained was based on European experience.

The position is now very different and as much is known about our sugar beet diseases as about the diseases of any crop grown in Britain. This is due in no small measure to the unremitting efforts and enthusiasm of Dr. Raymond Hull, who has devoted a quarter of a century to field and laboratory research on sugar beet diseases, first from the Midland Agricultural College, Sutton Bonington, and for the last ten years at the Dunholme Field Station near Lincoln, which for purposes of administration is attached to the Rothamsted Experimental Station.

In this revised edition Dr. Hull has summarized his knowledge and experience in a form designed to be of service not only to the plant pathologist but to all those engaged in growing or handling sugar beet.

Thanks are also due to the author's colleagues at Dunholme and at Harpenden who have advised on the experimental work which has produced much of the information in this Bulletin, the agricultural staff of the British Sugar Corporation for their help with surveys and field experiments, and the Sugar Beet Research and Education Committee who have financed and encouraged the work.

W. C. MOORE
Director, Plant Pathology Laboratory,
Harpenden, Herts.

November, 1959

Contents

Page

FOREWORD iii

SEEDLING DISEASES

Black Leg, Acid Soil Injury, Docking Disorder, Wind Injury, Frost Injury, Blind Seedlings, Strangles . 1

LEAF DISEASES

KEY TO YELLOWING 7

VIRUS DISEASES

Virus Yellows, 41-Yellows, Yellow Net, Mosaic, Dwarf, Beet Ringspot, Curly Top, Yellow Wilt, Leaf Curl 10

NUTRITIONAL DISEASES

Nitrogen Deficiency, Phosphate Deficiency, Potash Deficiency, Magnesium Deficiency, Manganese Deficiency, Manganese Toxicity, Boron Deficiency, Calcium Deficiency, Iron Deficiency . . . 20

MILDEWS

Downy Mildew, Powdery Mildew . . . 29

LEAF SPOTS

Rust, Cercospora Leaf Spot, Ramularia Leaf Spot, Pleospora Leaf Spot, Silver Leaf, Other Leaf Spots 33

VASCULAR DISEASES 36

WEATHER INJURY

Drought, Hail, Wind, Lightning . . . 37

CHEMICAL INJURY 39

INSECT INJURY

Aphids, Capsid Bugs, Frog-hoppers, Mites . . 39

GENETICAL ABNORMALITIES

Chimeras, Fasciation 41

ROOT DISEASES

Violet Root Rot, White Root Rot, Wet Rot, Scab, Girth Scab, Scurfy Root, Crown Gall, Crown Rot, Stem Rot, Fusarium Rot, Clamp Rot, Internal Fleck, Bacterial Soft Rot, Black Tail, Frost Injury, Lenticel Rot, Internal Zoned Rot, Eelworm Canker, Charcoal Rot, Dry Rot Canker, Southern Sclerotium Rot, Rhizopus Root Rot 41

Seedling Diseases

BLACK LEG

Black leg is a seedling disease in which the stem becomes discoloured and thin. The lesion varies from a superficial patch or streak to one completely encircling the stem, which is reduced to a slender, black thread and the seedling is eventually killed (Plate I, upper). Some seedlings may be killed before emerging from the soil. The disease is most serious on young seedlings and once secondary thickening has begun and the first true leaves have expanded, the plants are unlikely to be seriously affected.

Black leg occurs in every beet field but the losses are usually slight. It is of greater significance when efforts are made to obtain thin brairds by reducing seeding rate, using rubbed seed and other devices, in order to simplify the singling operation. Severe attacks sometimes occur in the south-west and west of Britain when the soil is unduly wet, and the disease is sometimes troublesome in other regions in crops sown early when the soil is cold or when other conditions are unfavourable for growth; for instance, when the seed bed is lumpy, too loose, or too firm, the soil infertile, the seed sown too deeply, or when the seedlings have been injured below ground by insects.

The disease is caused by various fungi which are normal soil inhabitants, and by one which is seed-borne. Of the soil-inhabiting fungi *Pythium ultimum* Trow and *P. intermedium* de Bary are the most common in eastern England. In other parts of the world different fungi are of greater importance; for instance, in North America the beet water mould *Aphanomyces cochlioides* Drechsler causes serious loss in soils of high moisture holding capacity. In this country, attack by *Pythium* is also favoured by wet soil conditions but is usually confined to very young seedlings. Although it is usually impossible to decide from a naked eye examination which particular fungus is responsible for black leg, the lesions caused by *Pythium* can sometimes be recognised by their water-soaked, purplish appearance. Such lesions develop rapidly and often kill the seedlings, leaving a withered hypocotyl with the cotyledons scarcely expanded.

Corticium solani (Prill. & Delacr.) Bourd. & Galz., often referred to by the name of the mycelial form, *Rhizoctonia solani* Kühn, is another soil-inhabiting fungus which attacks sugar beet seedlings. This fungus also attacks many other crops in cold, wet conditions, but there are several strains, each with its specific host range and not all of them attack sugar beet. Although the fungus may be found on occasional seedlings in most samples of black leg, severe attacks in sugar beet in this country are rare. When they do occur, patches of plants are attacked in otherwise healthy crops. The fungus affects both the roots and hypocotyl, killing some seedlings and leaving gaps in the braird. Some of the diseased seedlings recover by sending out fresh lateral roots above the lesion; such seedlings are usually smaller than healthy plants, giving an uneven braird often referred to as "Chicken and Hen". The British strain of *Corticium solani* which attacks sugar beet seedlings will not attack older plants, but in America there is a more virulent strain which

causes serious seedling losses and also produces a rot of fully grown roots (p. 52).

Pleospora betae Bjorling, previously known only in the imperfect stage, *Phoma betae*, is a seed-borne fungus which causes black leg and commonly attacks sugar beet plants at all stages of development. The lesions on young seedlings resemble those caused by parasitic soil fungi. *Pleospora* occurs more frequently than *Pythium* on older seedlings, but once secondary thickening of the hypocotyl has begun it is unlikely to cause further loss.

Seed infection with *P. betae* originates in the growing seed crop, because the fungus infects leaves and stalks causing leaf spots and stem rot, especially when the crop is ripening and the plants are becoming moribund. Spore cases (pycnidia) are produced on the affected tissues (Plate XV, upper right) and in damp conditions they exude a slimy, pink mass of spores which are disseminated by rain splashes. Conditions in stacked seed favour spread of the fungus, particularly if the crop is damp. The seed-coat then becomes infected with the fungus, which subsequently remains dormant until conditions are favourable for germination of the seedlings. Seed grown in humid conditions is often heavily infected. Infection is less in dry harvest years than in wet ones, and is lower in seed threshed and dried straight from the field than in seed which has been kept in stack for some time.

Pleospora betae produces a second form of fruiting body (perithecium), similar in appearance to a pycnidium, on the stems of infected seed plants and on pieces of infected sugar beet surviving the winter. The following summer these fruiting bodies liberate spores which can infect beet tissue. To this extent soil which contains buried stems may be infested and consequently cropping too often with beet or mangolds may lead to severe attacks. This is particularly so with seed crops, as many of the basal parts of stems left when the crop is harvested are infected and later worked into the soil. Similarly, sugar beet plants which have been affected with heart rot are often heavily infected with *P. betae* and if rotted plants and crowns are ploughed in, severe black leg is liable to develop in crops sown there the following year. Nevertheless, this is unlikely to happen in Great Britain, because factories will not accept contracts for sugar beet to be grown on land which has been cropped during the previous two years with sugar beet or mangold root or seed crops.

Black leg caused by *P. betae* can usually be controlled in this country by treating the seed with a fungicide, and all sugar beet seed supplied by the British sugar factories is so treated. The seed is agitated in a machine with an organo-mercury fungicidal powder to coat it evenly. Outbreaks of black leg are now rare, occurring only when the soil and weather do not favour seedling growth and the seed is too deeply infected with *P. betae* for the dry fungicide to reach it. *P. betae* is better controlled by soaking seed in a dilute solution of ethyl mercury phosphate (EMP) and then drying it, than by the powder, but this costs more and is used only on batches of seed sown at very low seed rates. Seed treatment with organo-mercury compounds also decreases the number of seedlings invaded by soil organisms, presumably by partially sterilizing a zone around the seed, but it is not effective when conditions greatly favour infection. Seedlings from treated seed grow more vigorously than those from untreated seed.

Growers have the choice of seed treated with a fungicide alone, or with fungicide and insecticide, usually *gamma*-BHC or dieldrin, to control both

black leg, wireworm and other insect pests in the soil. Seed dressings of *gamma*-BHC have sometimes injured seedlings as it is toxic to plants. Some polyploid varieties are particularly susceptible. The trouble is more common on light than heavy soils. It has largely been overcome by lowering the *gamma*-BHC content of the dressing and changing the formulation. Affected seedlings have narrow, thickened, distorted leaves with irregularly serrated edges, they are severely stunted and some die. The tap root is often discoloured or may be killed 1 to 2 in. below ground. When the plants are growing slowly the trouble may continue, with plants dying for a week or two after singling. Roots are fangy, stand far out of the ground and have a roughened surface.

When crops are so severely attacked by black leg that there are too few plants to give a reasonable braird, the only remedy is to plough and resow. Provided the seedbed is in a satisfactory physical state, the warmer weather at the later sowing usually results in a rapid growth of healthy seedlings.

Root development is encouraged by dressings of phosphatic fertilizers, and adequate dressings of superphosphate assist crops to resist and overcome attacks of black leg. When phosphate is lacking, seedlings grow slowly and many are soon killed by the fungi which cause black leg, as well as by others.

ACID SOIL INJURY

Acid soil injury is the most important seedling trouble of sugar beet in Great Britain and each year more seedling failures are due to this cause than to any other. Germination is often satisfactory in an acid soil, but the seedlings grow slowly and become smothered by the weeds characteristic of acid soils, e.g., spurrey (*Spergula arvensis*), sheep's sorrel (*Rumex acetosella*) and corn marigold (*Chrysanthemum segetum*). The cotyledons of seedlings in an acid soil are more erect than usual, and later the margins of the leaves often become red, or the plants may turn pale yellow as a result of manganese toxicity. Many plants die leaving a very gappy, uneven crop or a complete failure.

The primary roots of young seedlings in acid soils are curled and their tips die (Plate I, upper, *d* and *e*). Fresh lateral roots develop from the upper, healthy part of the primary root, but the tips of these also die and only a small proportion of seedlings survive. As a result of this type of root injury the plants produce masses of fibrous, curled rootlets which later develop into stunted and fangy sugar beet.

The symptoms on the hypocotyl differ somewhat from those of black leg and appear only when the first true leaves develop. Instead of increasing in thickness, the hypocotyl remains thin and stringy and has a characteristic scurfy brown surface, extending upwards to the cotyledons which sometimes wither (Plate I, lower left). Unless growth conditions are very favourable the plants develop slowly and many die. Those which survive may make reasonably good growth, but the scurfiness persists on the roots (see p. 45). Acid soil injury often occurs in patches which are easily recognised later in affected fields by the thin stand, the stunted plants and the fangy roots with masses of fibrous laterals, many of which are black and dead.

A number of fungi, particularly *Fusarium culmorum* (W.G. Sm.) Sacc. and *F. axysporum*, are associated with acid soil injury. The fungi which cause black leg also develop freely on plants growing in acid soil, so that confusion arises between the two diseases.

Although the trouble is primarily a result of soil acidity and in general becomes more severe with increasing acidity, its incidence is also affected very considerably by other soil factors, such as moisture content and fertility. Acid soil injury is particularly severe in dry soils, and a wet period may save a crop which appeared to be failing. Practices which conserve soil moisture and build up a high humus content may make all the difference between success and failure in a dry season. Damage may be negligible in a field of high fertility at an acidity which would result in failure in a field of lower fertility. It is not possible, therefore, to give a critical pH value above which the disease does not occur and below which failure is certain. It is unusual, however, for much damage to occur in soil with a pH above 6·4 and some loss of plants is almost certain to occur in soils with a pH of 5·8 or less; crop failure is highly probable in soils with a pH of 5·3 or less.

Complete control of the disease can be obtained by neutralizing the soil with lime. Soil analysis will indicate how much lime is required. It takes some time for lime to become thoroughly incorporated with the soil, and where severe acidity is likely to cause loss, lime should be applied at least in the autumn before sowing the sugar beet crop. Further information about liming is given in the Ministry's Bulletin No. 35.*

When the damage is not too great, it may be desirable to take measures to save a crop suffering from acid soil injury. Beneficial effects have been claimed from a top-dressing of 5 cwt per acre of hydrated lime well worked into the surface soil. Top dressing with 1–2 cwt per acre nitrate of soda or similar nitrogenous manure may also be helpful if an adequate amount of nitrogen has not already been applied with the fertilizer dressing.

DOCKING DISORDER

Poor growth of sugar beet in patches has been recorded since 1948 in the Docking area of Norfolk and recently elsewhere. Seedlings grow poorly despite adequate dressings of the usual fertilizers and normal cultivations because the tap root dies. Fresh laterals grow but they also soon die; this sequence is repeated as the soil is wetted by rain and dries again so that the stunted plants come to have many adventitious roots, most of which are dead. Some plants that partially recover have fangy tap roots with laterals growing horizontally a few inches under the soil surface. The root symptoms resemble acid soil injury but the soils on which Docking disorder occurs have a pH between 6·5 and 8·5, usually above pH 7. The leaves of affected beet are stunted and may show nitrogen or magnesium deficiency symptoms or both. The leaves of the worst affected plants are thickened and cupped. By August, patches of the field may be bare, and areas less severely affected have a thin stand of plants of very irregular size. The trouble occurs on the same patches each year, but may vary in severity. It is severe after wet, cold springs. With good growing weather affected crops may recover, but deteriorate quickly when the soil dries.

The soil in affected fields is fine sand, with a low clay and very low organic matter content and is alkaline. The trouble has arisen on this special type of soil with farming practices which have depleted soil organic matter

* Bulletin No. 35, *The Use of Lime in Agriculture*. Price 3*s*. 6*d*. (by post 3*s*. 11*d*.). Obtainable from H.M. Stationery Office or through any bookseller.

— no livestock, no leys, frequent cereal crops cleaned with chemical weed-killers and long autumn fallows, burning straw and deep ploughing.

Although the scientific explanation of the cause of the trouble has not been determined, several ways in which it can be overcome are known. After several years under lucerne the land grows good sugar beet crops. Crops respond to heavy dressings of farmyard manure when they will not respond to chemical fertilizer. Dressings of shoddy have a big effect on growth of beet. Soil treatment with the sterilant DD strikingly improves the growth of beet; this does not seem to result from killing a parasitic organism because beets fail to respond when the soil is sterilized by steam.

WIND INJURY

Sugar beet seedlings are liable to suffer severely from wind injury on light soils subject to wind-erosion, by being buried in blown sand or having the soil blown away from their roots. They also suffer in other ways; at the cotyledon and first leaf stages the overground parts may be killed by the sand-blasting effect of blown soil particles, or the hypocotyl may be damaged at soil level, producing an injury which later develops into strangles.

This is not the place to deal with the subject of soil erosion, but some ways of preventing it in sugar beet fields may be mentioned. "Blowing" is particularly severe in fields which are left rolled after sowing, whilst those which are harrowed are not affected to the same extent. The best procedure is to drill after rolling, using a drill with rolls fitted behind the coulters, and then cross-harrow after drilling. The problem has been overcome on black fen soil by preparing the seedbed in February, cultivating 12 in. bands along which to sow the beet in early April and applying pre-emergence weed-killers along these bands before the seedlings emerge. The soil adjacent to the beet rows is kept free from weeds by steerage hoeing with 4 in.-cut blades, but the weeds are not disturbed in the centres of the rows until June when the risk of "blowing" is over. They may be then about a foot high and are cleaned up with a rotary cultivator.

Some growers sow beet between rows of barley which is later hoed out. Others find the loss can be prevented by making a furrow in between each row with a spring-tined implement at the first sign of blowing, treating every alternate row is often enough. Should the wind blow along the rows, then the tines are taken across the rows; a few plants are lost but the crop may be saved.

FROST INJURY

In general, sugar beet seedlings are capable of surviving the degree of frost normally encountered in April and May, but frost injury sometimes occurs in fields in which the seedlings have only just emerged, or in districts liable to severe ground frosts. Fens and low lying fields are usually most severely affected. Severely frosted seedlings have a flaccid, water-soaked appearance, and quickly wither and die on thawing, whilst the underground parts remain sound for a few days. If the frost has not been lethal, the cotyledons are thickened and curled and usually have a silvery appearance, and the minute young leaves around the growing point are blackened.

BLIND SEEDLINGS

Blind seedlings are those in which the primary growing point has been killed. The commonest cause is a capsid bug which feeds and lays its eggs around the growing point. If not killed, the minute young leaves are distorted and pink. Stem eelworm causes similar injury (see also p. 51). These pests are more fully described in Bulletin 162.* Blind seedlings may also be caused by the toxic effects of *gamma*-BHC seed dressing or by mechanical injury or by frost.

STRANGLES

When singled plants are growing rapidly in May or June, the overground parts sometimes break off at soil level when disturbed by cultivation, or by wind. This fragility results from a deep constriction of the tissues at or about soil level. The shoulders of the constriction are rounded and smooth, and the injury is obviously not the result of recent insect attack (Plate I, lower right). A black, sugary, sticky fluid often exudes from the constriction. Before the top breaks off affected plants may look healthy, but occasionally they are stunted or yellow. The trouble, known as strangles, occurs commonly in a small proportion of plants in fields all over Britain. The trouble is most prevalent in Scotland and the west of England where losses of 10–20 per cent are common and sometimes as many as 50 per cent of plants may be lost.

Strangles results from an early injury to the hypocotyl from which the plants do not completely recover. The tissues above and below the injured part continue to grow and increase in diameter, but the two regions usually do not fuse. The upper part of the plant is connected to the root only by a narrow strand of vascular tissue which is continually weakened as the top is flexed by the wind. In some affected plants the tissues may fuse and then the constriction is gradually healed. Other plants show two constrictions, the hypocotyl between them being normally thickened. There may also be long-necked constrictions, or only one side of the root may be affected.

The cause of strangles is not definitely known, and the initial injury to the unthickened hypocotyl may have several causes. Black leg, acid soil injury, wind damage and insect attack are all possible primary causes, but as strangles occurs only in singled crops, exposure of the hypocotyl to wind and sun, by removing excessive amounts of soil during gapping and singling, may be the most important form of injury. Experiments have shown that strangles is more prevalent in crops sown on ridges than on the flat. High seeding rates produce "drawn" seedlings which increases the proportion of strangles, and fewer plants have strangles in crops singled at the four leaf stage than in crops singled earlier. Strangles is worse on fields with a hard soil surface than where the tilth is loose. Thus its incidence can be lowered by level seed beds, low seeding rates, late singling, not exposing the plants too much when singling and maintaining a good tilth.

Once strangles occurs in a field of singled plants, there is little that can be done, but working loose soil up to the plants sometimes helps plants to recover. The grower need not be unduly alarmed when he notices this trouble, for it will not spread to other plants, and it usually does little more than decrease plant population slightly.

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* Bulletin 162, *Sugar Beet Pests*. Price 6*s*. 6*d*. (by post 6*s*. 11*d*.). Obtainable from H.M. Stationery Office or through any bookseller.

Leaf Diseases

THE RESPONSE of sugar beet to numerous diseases and adverse factors is for the foliage to turn yellow and later become necrotic. Because the individual plants in a beet crop are of diverse hereditary constitution (for sugar beet seed can only be produced by cross-fertilization) they react differently to disease infection. Thus a single disease may show a range of symptoms on different plants in a field, and since the ranges for different diseases overlap, confusion is easy. When diagnosing the cause of yellowing in sugar beet, it is necessary to take into consideration details of shade, pattern and texture, the circumstances in which the plants are grown, and the range of symptoms on different plants. The characters of each disease are described in detail in ensuing sections; it may be helpful at this point, however, to consider the yellowing diseases as a group and to differentiate between them (*a*) according to the time of year when they are likely to be encountered in the field, and (*b*) according to the symptoms.

KEY TO YELLOWING

JUNE

Yellowing of patches of plants is most likely to be due to nitrogen, iron or manganese deficiency. In some seasons magnesium deficiency may occur. Soil acidity results in patches of stunted plants and when associated with manganese toxicity they are markedly yellow. Occasional plants may be infected with virus yellows and mosaic, and the outer leaves of plants attacked by downy mildew become chlorotic. Yellowing of leaf tips associated with distortion and blisters on the veins and midrib is due to capsid bug injury and usually develops on plants near hedgerows and woods. Occasionally plants look yellow and stunted because of strangles or insect injury to the tap root. Circular patches of stunted plants may be produced by lightning injury.

JULY AND EARLY AUGUST

The same diseases are encountered as in June. Yellowing due to downy mildew is likely to increase and early signs of magnesium, potash and boron deficiency may make their appearance. Spread of virus yellows from the early centres of infection results in rounded patches of plants with yellow outer leaves. Chimeras become obvious.

LATE AUGUST AND SEPTEMBER

The symptoms of iron and manganese deficiency and yellowing due to downy mildew is less obvious. Virus yellows increases and the outer leaves of plants infected earlier become necrotic. Yellowing due to magnesium deficiency also increases and both the older and younger leaves develop necrotic spots and margins. Boron deficiency will increase and in dry weather may lead to much yellowing and death of the foliage, and may kill patches of plants. Vascular diseases are likely to show up as well as those root diseases which are accompanied by symptoms on the overground parts. Wilting, yellowing and

necrosis resulting from drought may become serious in hot, dry summers and mites may develop during hot, dry spells, resulting in yellow blotches on the leaves. Rust, leaf spots and powdery mildew may become so severe that they cause yellowing and browning of the older leaves.

October

The symptoms of deficiency diseases usually tend to decrease in intensity. Virus yellows infection continues to increase, but the intensity of yellowing may be somewhat masked by new growth of the central leaves.

Symptoms

Roots abnormal

Examination of the roots of a diseased plant will indicate whether yellowing of the foliage is the result of injury or disease below ground. Lightning injury, insect injury, wet rot, strangles and violet root rot at times all result in yellowing and necrosis of the foliage. Pythium yellows, bacterial rots and internal zoned rot, which also give the foliage an unhealthy appearance, may be recognized when the roots are cut across — Pythium by discoloration of the vascular strands; bacterial rots by discoloration, smell and slime; and internal zoned rot by the discoloration and decay of the interior of the root.

Stunting and necrosis of the younger, and yellowing of the older leaves

If the younger leaves are stunted but not necrotic, nitrogen deficiency is probably the cause. The leaves of plants infected with downy mildew are at first thickened and distorted and later become necrotic. At the latter stage the symptoms are similar to those of plants suffering from heart rot, which can be distinguished, however, by a discoloration between the outer vascular rings of the root and by dry rot around the crown. Other symptoms characteristic of heart rot are the formation of brown scales in the lower part of the groove on the upper side of the petiole, the development of cracks on the laminae of the oldest leaves and the prostrate habit of the plant. Also, in heart rot the growing point and youngest leaves of the rosette are the first to die, whereas in downy mildew traces of living, infected tissue often remain in this region. In autumn the edges of the young leaves of plants suffering from magnesium deficiency often become necrotic, but in addition there are always typical chlorotic symptoms apparent on the older leaves of some affected plants. Severe attacks of black aphid result in much puckering and browning of the foliage but even when the aphids have gone, their depredations can be recognized by the grey cast skins and honeydew on the leaves.

Yellowing and necrosis of the older leaves; young leaves normal

Three diseases in this category are likely to be confused — yellowing of the older leaves following downy mildew attack, virus yellows and magnesium deficiency. The leaf symptoms of magnesium deficiency may be distinguished from those of the other two by the chlorotic pattern resulting from the sharp demarcation of the yellow interveinal areas from the green areas around the veins; by a dark brown or black necrosis, often restricted at first to the margins of the leaf; by the ragged margins of affected leaves resulting from the disintegration of necrotic tissue and, later, by the dark coloured necrosis

of the margins of the younger leaves. Virus yellows and downy mildew both cause the outer leaves to become thickened and brittle and the yellow and green tissues merge into one another so that there is no definite pattern of yellowing. Distortion, thickening and necrosis of the younger leaves indicate that downy mildew may be the cause of yellowing of the outer leaves, but without laboratory tests it is not possible to say whether or not they are also infected with virus yellows. Discoloration of the vascular tissue in the petiole, sugary exudations on the surface, and a purplish brown necrosis spreading from the petiole and veins into the leaf-blade are indications of downy mildew. Clearing of the veins of the younger, and etching of the veins of the older leaves, brown freckles, and reddening are characteristic of virus yellows.

Dull, yellow blotches and bronzing around the margins of the leaves, associated with a rapid development of lobes of light brown necrosis between the veins, are signs of potash deficiency. The foliage is likely to be lush, dull and dark green. The leaf diseases caused by fungi such as rust, cercospora and ramularia leaf spots, also produce some yellowing and necrosis of the older leaves, but the lesions of leaf spot diseases can be recognized on the yellow and necrotic tissue as well as on the younger, less severely damaged leaves. The white mycelium of powdery mildew is also visible on the leaves which it kills as well as on the younger leaves. The yellowing, necrosis and spotting produced on the outer leaves which wilt during drought, are often difficult to diagnose with certainty when the plants have recovered somewhat after rain, but there may be bleached, necrotic leaves lying on the ground, though still attached to the crown; the nature of the soil and subsoil are also an indication whether severe wilting is likely to have occurred. Yellowing due to capsid bug injury can be recognized by the warts on the veins and distortion of the leaf. Yellow interveinal patches which develop on the older leaves late in the summer during hot, dry periods may be due to attack by mites.

Mottling of the leaves

The most common cause of mottled sugar beet leaves is manganese deficiency. Severe cases are easily recognized, not only by the characteristic starry interveinal yellowing, but by the upright, staring habit of the plants, and by the incurled leaf margins which give the leaves a triangular appearance resembling a hare's ear. Plants which have recovered from the more severe symptoms may be recognized by the angular, necrotic lesions on the older leaves and a faint mottle on the younger. The disease may be confused with mosaic, but an examination of the leaf against the light will show that the finer grained mottle of mosaic results from the veins being narrowly banded by dark green tissue, or from light green rings with a point of dark tissue in the centre. No obvious stunting or necrosis occurs in mosaic, and by the lack of these characters it may be distinguished from dwarf due to cucumber mosaic virus. It is often difficult to distinguish between manganese deficiency and iron deficiency, which also produces an interveinal mottling, but the mottle of iron deficiency is usually finer than that of manganese deficiency, and in severe attacks the whole leaf is a bright, light yellow with the network of veins clearly picked out in green. The yellow blotches caused by mites sometimes give the leaves a mottled appearance, but this trouble can readily be recognized by the presence of the insects on the underside of the leaf and by the web they produce.

General yellowing of the foliage

Nitrogen, manganese and iron deficiencies in their most severe forms produce a general yellowing of the foliage, but there are always some plants or patches in a field showing less intense symptoms which are characteristic of the particular trouble. The most common cause of patches of plants turning yellow in the early summer is manganese toxicity, associated with acid soil conditions. Individual plants which are wholly or partly yellow, light green or white are usually chimeras or other genetical sports.

Necrosis of leaves with negligible yellowing

Phosphate deficiency results in dark brown or black necrotic marginal lesions with lobes extending between the veins of otherwise green leaves. The affected plants are stunted and the crown is long-necked with many dead leaves around it. Calcium deficiency causes the leaf blades to be attenuated. The tips and edges are necrotic and cease to grow, and as the middle of the leaf continues to grow for a time, the leaf blade becomes concave. In more severe attacks the lamina of the leaf is reduced to a black tip on the end of a stunted leaf stalk. Both magnesium deficiency and potash deficiency may result in necrosis with negligible preliminary yellowing, but plants with more distinctive symptoms can usually be found and these enable the disease to be correctly diagnosed. Rounded, blackish or dark brown lesions on green tissue, zoned into light and dark bands, are due to pleospora leaf spot.

VIRUS DISEASES

VIRUS YELLOWS

Symptoms

The first symptom of virus yellows noticed in the field is a yellowing of the tips of the middle-aged leaves. Gradually the whole leaf becomes yellow, although the tissues along the veins remain green for some time. The yellow and green tissues merge into each other, and are not so sharply defined as in magnesium deficiency (Plate VII, upper and lower). The yellow leaves are thick and brittle and when crushed in the hand they crackle and break into pieces. Infected plants wilt less easily than healthy ones and on bright, hot days they may remain upright when healthy ones are wilting. The yellow areas become necrotic, first at the tips and edges of the leaves, and later in lobes extending between the veins. The necrotic leaves remain entire, though they may eventually fold up, the edges and tip rolling inwards. As infection progresses, all the older leaves become chlorotic and finally necrotic (Plate V, upper). The youngest leaves usually remain green, and if soil moisture and nutrients are adequate, they may continue to make fresh growth, thus tending to mask the yellowing of the outer leaves. In June it takes ten to fourteen days for the symptoms to appear on young plants after they have been infected by aphids, but it may take four to six weeks on older plants or in dull weather. The yellowing on the leaves of older plants infected later in the season may be localized on part of the leaf, often in an area bounded on one or two sides by a vein; such plants are not obviously stunted and there is usually no necrosis.

The symptoms developed by individual plants may vary. Some plants first show a superficial necrosis of the veins at the leaf tips, giving them an etched appearance which may persist throughout their life. On other plants the veins of the youngest leaves become glassy and light yellow, a symptom known as vein clearing; this condition is usually ephemeral but occasionally persists. Some strains of the virus, usually the more virulent ones, are more prone to produce these symptoms of vein clearing and vein etching than the milder ones. Many infected plants develop brown freckles on the chlorotic tissues, and the leaves of others may become scarlet instead of the more usual bright yellow or orange yellow.

The necrotic stage is associated with attack by a number of weakly parasitic fungi, the most common of which are *Alternaria tenuis* and *Sporodesmium putrefaciens:* they form spores freely on the surface of the necrotic tissues. Various forms of necrotic lesions and isolated spots, often resembling true leaf spot diseases, may be produced by these fungi on yellowing leaves. Some plants develop small, circular red spots with a minute necrotic centre. Rust (*Uromyces betae*) and cercospora leaf spot are often more prevalent on plants with yellows than on healthy ones.

Virus yellows does not usually appear in the root crop until June or early July, when infected plants may be found scattered indiscriminately throughout the crop. Later, the spread of the disease first becomes apparent as a general paling of the crop in patches. The extent to which the disease spreads is very variable. Sometimes large patches, or even the whole crop, may show signs of infection by early August. At other times the spread is so slow that by the end of September there are still only a few patches of infected plants. If infection is widespread in August the crop assumes a bright yellow colour and is so stunted that the soil can be seen between the rows, and excessive weed growth occurs.

The symptoms of virus yellows on seed plants are essentially similar to those on the root crop (Plate V, lower). In summer and autumn, infected stecklings develop typical yellowing, but as symptoms are rather slow to appear in autumn, many plants which seem to be healthy are, in fact, infected. After the older leaves have been killed by winter frosts it is often impossible to recognize the symptoms in plants which were undoubtedly infected earlier. The following spring infected stecklings sometimes show rather indefinite symptoms on the oldest leaves in May, but generally the new spring growth appears to be healthy at first and does not become yellow until June or July. After this it rapidly becomes necrotic. Infected plants are stunted to varying degree and this probably depends on the date of infection. Seed plants which are infected in spring turn yellow much sooner than those infected in the previous autumn, but they are not so severely stunted. Plants infected with virus yellows are usually ready for harvesting about two weeks before the healthy ones.

Effect on Yield

Virus yellows is the most serious disease of sugar beet in Great Britain and occurs in all parts of the country. The most widespread and severe attacks have been experienced in the eastern parts of England, and although in the south, west and north local severe outbreaks occur sporadically, attacks are generally less severe, and in some years may be negligible.

In years of severe attack the average yield of roots for the country may

be reduced by two or more tons per acre. The loss of yield depends largely on the date of infection. In crops which are infected at the end of July or early in August yields may be reduced by about 50 per cent. Later infections have a correspondingly smaller effect on yield. Sugar content may be reduced by 1–2 per cent and the processing quality of the roots is adversely affected. From the results of field experiments carried out over a number of years it has been calculated that for every week a plant shows symptoms of virus yellows up to the middle of October, it loses 4–5 per cent of its potential yield of sugar. Therefore, if the percentage of plants that are infected at intervals during the season is known, it is possible to estimate the loss of sugar yield per acre caused by the disease.

The effect of the disease on the yield of sugar beet seed crops has not been so accurately determined, but in some experiments the yield of seed was reduced by 30–50 per cent. Moreover, the seed from infected plants is smaller and has a poorer germination capacity than that from healthy plants.

Sources of Infection

Beet yellows virus is spread by aphids, which may continue to infect plants for several days after they have become infective by feeding on a diseased plant. The severity of attacks of virus yellows in beet crops depends, therefore, upon the extent of sources of virus and the numbers and behaviour of the aphid vectors. The virus is not seedborne, so that the young root crop begins its life free from the disease each year. The virus is carried to it by winged aphids from live plants of the beet family which become infected in the previous season and survive over winter. The seed crops of sugar beet, mangolds and red beet, the stecklings of which become infected in the autumn, are important sources of the virus in the following year. The disease also spreads from mangold and red beet clamps, for not only do the sprouts of the clamped roots provide a source of virus, but virus-carrying aphids (e.g., *Myzus persicae*) may survive the winter on them. Other sources from which the virus may be spread in the spring are garden crops of winter spinach and seakale beet; badly topped sugar beet and small unharvested roots which grow up in the following crops; and wild beet plants (*Beta maritima*) which grow in profusion on the seashore banks on parts of the coasts of east, south and western England. Several weeds such as goosefoot (*Chenopodium album*), groundsel (*Senecio vulgaris*), chickweed (*Stellaria media*) and others, are susceptible to infection by the virus. Most are annuals, and thus play no part in maintaining the annual cycle of infection overwinter. Others undoubtedly live overwinter, especially in sheltered places, and may, in some years, be sources from which the disease spreads to young root crops.

Vectors (insect carriers)

The peach-potato aphid, *Myzus persicae* Sulz., is the most important carrier of the beet yellows virus. It is an efficient vector, a single aphid being capable of transmitting infection to several plants. The winged forms are very active and may feed for a short time on many plants. During mild winters the insects persist in large numbers, not only on their chief winter hosts (savoy cabbages, turnips and other brassicas) but also on plants of the beet family such as sprouts of clamped mangolds and stecklings. Migrants from the two last-named sources are the most dangerous because they carry the

yellows virus if the plants on which they fed were infected, whereas brassica crops do not harbour this virus. In severe winters the peach-potato aphid is more dependent for its survival overwinter on the eggs laid on peach trees, but it may also overwinter in mangold clamps, enclosed gardens, glasshouses and other sheltered places. The life cycle of *M. persicae* is illustrated in Fig. 1 (p. 14).

The black bean aphid, *Aphis fabae* Scop., sometimes infests sugar beet crops very heavily. It is a vector of beet yellows virus and spreads the infection introduced into the root crop by *Myzus persicae*. It is possible, also, that the migrants from the heavy infestations of black aphid which sometimes develop on sugar beet and mangold seed crops may carry the virus and cause widespread late infection in the root crop. *Aphis fabae* can survive the winter only as eggs, which are laid on spindle bushes (*Euonymus europaeus*) and the snowball tree (*Viburnum opulus sterile*). Its life cycle is illustrated in Fig. 2 (p. 14).

The aphid population which develops in early summer is dependent to a large extent on the number of aphids surviving overwinter and later developing in early spring on the winter hosts. If large numbers of *Myzus persicae* survive overwinter on brassica crops, they are able to build up large populations of migrants by the time they move to sugar beet; when this happens virus attack is usually early and severe. The autumn weather, the extent of the rise and fall of aphid infestation, and the profusion of their parasites (e.g. ladybirds, hoverflies and minute wasps) during the previous summer, determine the size of the populations at the beginning of winter. After severe winters the development of colonies from eggs and green aphids surviving in sheltered places becomes of greater importance, and this depends not only on the number of eggs laid on the winter hosts in the autumn, but also on the weather in the following spring. Early springs with dry, warm, calm weather favour aphid development and movement: cold, wet, windy conditions restrict them. Severe virus attacks are associated with mild winters and dry, hot springs and summers.

Control

As with all infectious diseases, the first step towards control is to eliminate sources of infection. As the virus is carried by flying aphids it may be spread over considerable distances, and although measures taken to eliminate sources of infection on any one farm may reduce the chances of severe attacks, they will not prevent infection coming in from elsewhere. But effective control measures, exercised on a large scale, are likely to have a cumulative effect because, if sugar beet and related root and seed crops can be kept healthy in one year, the chances of a severe outbreak of the disease in the following year are greatly reduced.

Control in the seed crop is most important, not only because it increases the yield of seed, but because of the importance of the seed crop as a source of infection for the root crop. Healthy seed crops may be obtained by planting healthy stecklings. All steckling beds are now inspected in the autumn. Those certified as having less than 1 per cent of plants with yellows are suitable for planting. Stecklings raised in root and seed growing areas are often heavily infected in the autumn, but it is possible to raise healthy plants in other districts and plant them out in the present seed growing areas. The most suitable areas for raising stecklings are those where pasture or cereal

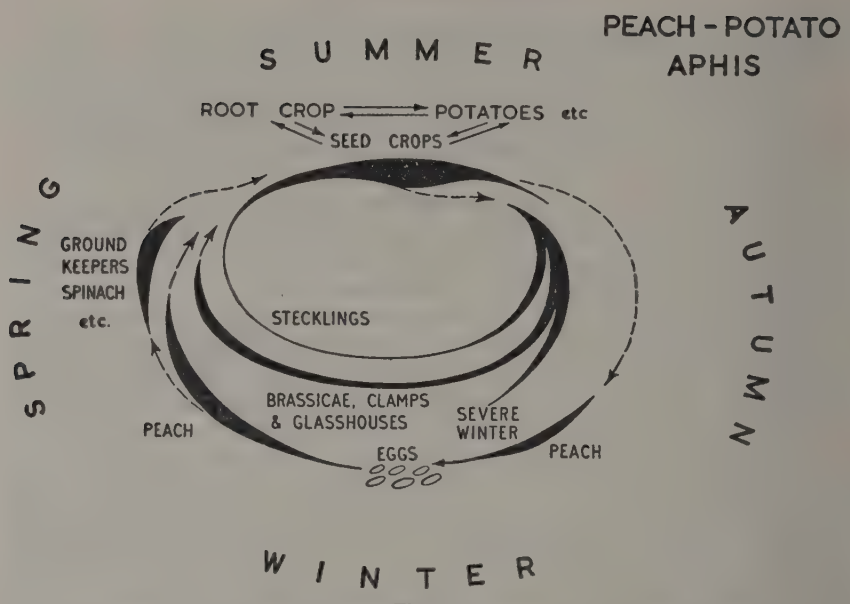


Fig. 1

Diagram illustrating the life cycle of the peach-potato aphid (*Myzus persicae*). The thickness of the solid lines represents the size of the population, and the dotted lines and arrows represent movement of winged forms.

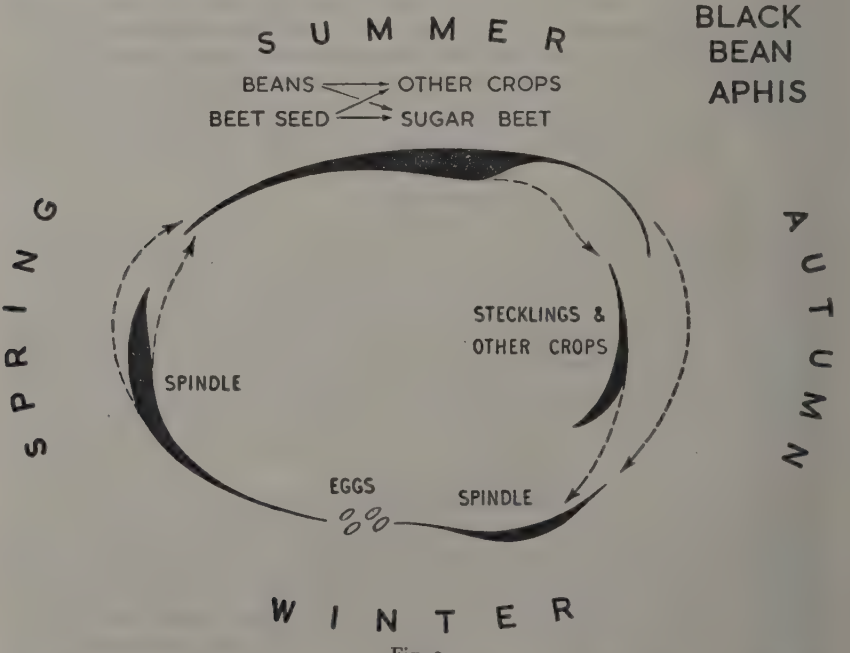


Fig. 2

Diagram illustrating the life cycle of the black bean aphid (*Aphis fabae*). The thickness of the solid lines represents the size of the population, and the dotted lines and arrows represent movement of winged forms.

crops are chiefly grown, with few mangold, sugar beet or market garden crops.

Less infection occurs in late- than in early-sown stecklings. If the seed is sown on moist, fertile soil, good plants may be obtained from sowings as late as mid-August. Infection may be further reduced by spraying the young stecklings with systemic insecticide, which kills the aphids. The plants should be sprayed at two-weekly intervals throughout the autumn. The most effective application is that put on about the middle or end of October; this kills the aphids at a time when re-infestation has ceased, and consequently the steckling bed remains free from aphids overwinter. Late sowing and spraying with systemic insecticide may result in the production of healthy stecklings in marginal districts where neither sugar beet nor mangold root and seed crops are grown intensively, but it is unlikely that such control measures would regularly ensure the production of healthy stecklings in intensive seed- and root-growing areas.

Yellows has been most consistently controlled by raising stecklings under cover crops. Either the stecklings are drilled across the rows of a spring cereal crop, sowing the two crops simultaneously in April, or every third coulter is blocked off in the corn-drill and the stecklings are sown along the blank rows when the corn emerges. The stecklings make little growth until the cereal is harvested. The cereal crop must not be so thick as to smother the stecklings. They do not survive under lodged crops. The stecklings need spraying with insecticide to protect them against aphids after the cereal has been harvested. They may be transplanted or grown-on *in situ* to produce seed.

Whenever possible, all mangolds should be fed to stock before the beginning of April, the clamp debris burnt and the clamp site ploughed. If mangolds must be kept for later feeding a special clamp of closely topped roots should be made for the purpose. The most heavily aphid-infested clamps are those containing untopped mangolds, for *M. persicae* are taken into the clamp in the autumn on the leaves. When topping to avoid this the roots must not be cut below the lowest leaf scars and bud initials or they will not keep. Clamps made from closely topped roots usually remain free from *M. persicae*, and the roots free from sprouts. They are cooler and may need heavier protection in cold weather than clamps of untopped roots. The mangold clamp aphid, *Hyperomyzius staphyleae*, may be introduced into clamps on roots and soil, but it is a less important vector of yellows than *M. persicae*.

Measures taken to control yellows in mangold crops, such as early sowing, high plant population and spraying with insecticide will improve the yield of mangolds and decrease the incidence of infective aphids in the clamps.

Small lots of mangolds infested with aphids in the spring should be used immediately. Large clamps are more difficult to deal with. Either they may be covered closely and any shoots pushing through the surface sprayed with insecticide; or the straw covering may be loosened and fired. This spoils the surface mangolds but aphids do not thrive on the remaining mangolds as well as when they are covered. The roots should be used as quickly as possible.

Although virus infection may spread from ground-keeping beets, they are not an important source of infection because they do not normally become infested with aphids when growing in cover crops. But their presence is

undesirable from the point of view of other pests and diseases, and measures taken to avoid them are well justified. The number found in following crops depends on the efficiency of harvesting: it may be reduced by close folding sheep on the tops and by deep ploughing. Weed-killers applied to cereal crops do not usually kill them, but cause them to shrivel up sufficiently to ensure that they are not infested with aphids before the corn outgrows them.

Seakale beet, commonly grown in gardens, is often infected with yellows and often heavily infested with *M. persicae* in spring. It is an important source of virus-carrying aphids for crops near urban areas, but there is little a farmer can do about it.

The incidence of yellows can be lowered by spraying root crops with persistent, systemic insecticide to kill aphid vectors and ensure that they do not colonise the plants for a few weeks. Spraying does not prevent winged aphids infecting plants with yellows on which they feed when they first reach the crop, but soon kills them and prevents them from multiplying and spreading the disease within the crop — hence the importance of eliminating sources of infective aphids.

The most effective sprays found so far are organo-phosphorus systemic insecticides of which there are several proprietary brands. Recently, fluoro-acetamide has also been used. All these materials are poisonous to human beings and livestock and the precautions advised by the makers should be carefully followed.

Growers whose crops are regularly infected with yellows, particularly those in the south-east of England, should give a routine spray. This should usually be in mid to late May after singling, when the plants are the size of saucers. It depends, however, on the time of aphid invasion, which after mild winters and early springs may be while the crop is still in braird; after hard winters and late springs not until the middle or end of June. A second spray two to three weeks later usually pays. In other areas where yellows occurs more sporadically, spraying should be delayed until a few green aphids (an average of one aphid on two plants) can be found on the underside of the leaves. The British Sugar Corporation agricultural staff have recently surveyed crops for aphids during the spring and warned growers when to start spraying. The yield increase obtained by spraying when yellows is prevalent may be as much as 4–5 ton per acre of roots and 1–2 per cent sugar content. In most years it is less spectacular, but economic responses are obtained when about 20 per cent of plants have yellows on unsprayed crops at the end of August.

Early-sown crops usually contract less infection than late-sown crops, and the disease occurs at a later stage of development of the plant, so that there is a smaller percentage loss of yield in early-sown crops. Fields with high plant populations tend to be less heavily infested with aphids than those with thin, gappy stands. and consequently they are less heavily infected with virus. Some varieties of sugar beet are slightly more tolerant than others but the difference is not great enough in commercial varieties to justify the choice of any one solely for this reason. Variations in fertilizer treatment have very little effect on the spread of infection or on loss of yield. Although heavy dressings of nitrogenous fertilizer may give a crop a greener and healthier appearance, they reduce neither the percentage of infection nor the loss caused by infection.

41-YELLOWS

The virus disease, 41-yellows, has occurred only rarely and only in experimental breeding lines of sugar beet. It can be transmitted from one sugar beet to another with difficulty by *Myzus persicae*. It is seed-borne and as many as 30–40 per cent of plants growing from infected seed may show symptoms within three or four weeks of germinating. The symptoms resemble those of virus yellows but the yellowing is paler, and more diffuse, and reducing sugars do not accumulate in the leaves of infected plants as with virus yellows. This disease does not occur in commercial crops in Great Britain.

YELLOW NET

Yellow net is a virus disease, transmitted by *Myzus persicae*, which occurs sporadically in the United States and rarely in Britain and Europe. The network of leaf veins becomes bright yellow; and severely affected leaves become pale yellow all over. Sometimes the veins of only half or part of a leaf are affected.

MOSAIC

Plants infected with beet mosaic virus are not usually stunted or greatly discoloured; the leaves merely show a fine mottle of light and dark green (Plate IX, lower). The mottle may consist of dark green bands along the veins with lighter tissue in between, or small light green rings on a darker background. These symptoms are best seen by transmitted light. They are most pronounced on the younger leaves and may fade on older leaves. The veins of the young leaves are "cleared" or yellow at first, but this does not last long. There is no leaf necrosis.

Symptoms of mosaic are most pronounced on stecklings in the autumn and spring and infected seed plants are difficult to recognize in July. Plants infected with both yellows and mosaic viruses (Plate V, lower) show symptoms of yellows on the older leaves, mosaic on the younger and symptoms of both diseases on intermediate leaves. Seed plants infected with both diseases are often obviously stunted.

Cultivated plants which are closely related to sugar beet, such as red beet, mangold, and spinach, and also a number of weeds, may be infected with beet mosaic virus. Infection of the common weed goosefoot (*Chenopodium album*) causes severe distortion and necrosis and usually proves fatal, but others, such as chickweed (*Stellaria media*) show no symptoms.

Mosaic spreads in the same manner as virus yellows. The same sources of infection and the same vectors are involved; but there is an essential difference in the nature of the viruses and their relationship to the vectors. An aphid which has fed on a yellows-infected plant can for several days infect healthy plants on which it feeds, but one which has fed on a mosaic-infected plant is infective for less than one hour. Consequently, mosaic in sugar beet crops tends to be more localised around the main sources of infection than yellows. Mosaic virus can be transmitted from plant to plant mechanically; for instance, by rubbing healthy leaves with the sap of infected plants. It is

possible that the disease is spread in this way in seed fields when the leaders are cut or pinched out in spring.

Mosaic is not a serious disease in Great Britain. Severe attacks are usually accompanied by virus yellows and losses from mosaic are small compared with those from yellows. Mosaic is prevalent only around local infection sources, of which the most common are clamped mangolds and wild beet. Seed crops, and root crops near them, used to be heavily infected, but the measures taken to control yellows in steckling beds have also controlled mosaic.

Control measures are essentially similar to those for virus yellows; to raise healthy stecklings and thus ensure mosaic-free seed crops; to keep seed crops, root crops and steckling beds separated as far as possible; and to clear up sources of infection, such as mangold clamps and groundkeepers. Where infection sources cannot be eliminated, as in coastal areas where wild beet is prevalent, spraying root crops in June with systemic insecticides to control yellows will also decrease mosaic.

DWARF

Dwarf is the name given to a disease of the seed crop caused by cucumber mosaic virus. Infected plants may be found in the steckling bed in autumn, usually in groups occupying short lengths of row. The leaves are very narrow and stunted, frilled at the edges, twisted and curled, often with the edges rolled underneath. At first the leaves are light green with a chlorosis along the veins but later a yellowish-green mottle develops. After this they become slightly bronzed, with a dull, etched appearance. Some of the most stunted leaves may have black, necrotic tips, making them pointed. The outer leaves often die, though they remain attached to the plant. The roots are poorly developed and have few living laterals, most being blackened or dead. Some infected plants die; the less severely affected ones, which survive to be planted out in the following season, produce plants showing similar foliage symptoms to the stecklings, but remain dwarfed, growing to a height of 6–12 in. as compared with healthy plants of 3–4 ft (Plate II, lower). In some plants the main and lateral growing points die backwards from the tips, but in others an inflorescence with few minute seeds is produced.

The peach-potato aphid (*Myzus persicae*) is probably the most important vector of this virus in the field. Cucumber mosaic virus can infect a wide range of plants, including cultivated crops such as cucumber, vegetable marrow and tomatoes, and hedgerow plants such as white bryony (*Bryonia dioica*), which is commonly infected.

The disease has not yet been prevalent enough in sugar beet crops to cause serious loss but its effect is so drastic that appreciable attacks might prove serious. A small number of infected plants is sometimes found in commercial sugar beet seed crops, but the most severe infections, usually about 10 per cent (and 50 per cent in one instance) have occurred in stecklings raised for experimental purposes in gardens. Presumably infection spreads from nearby infected weeds, such as white bryony, and control lies in eliminating these weeds and avoiding sites for steckling beds which, from experience, are known to produce infected plants. Measures taken to control yellows and mosaic will also control dwarf and other aphid-borne virus diseases.

BEET RINGSPOT

Beet ringspot is caused by a soil-borne virus — a strain of tomato black ring virus. It has been found in a few localities in Scotland, often in association with the soil-borne raspberry ring spot virus. It occurs in patches in soils of light texture and infects turnip, swede, potato and many kinds of weed as well as sugar beet. The virus infects through the roots and later spreads to the leaves, which may remain symptomless or may develop chlorotic tissue in an irregular ring pattern. The economic importance of the disease is not known.

CURLY TOP

Curly top is a very severe disease of sugar beet in the arid and semi-arid regions of western North America; it has recently been found on sugar beet in Turkey, but does not occur in Britain. Before control measures were developed, its effects were so serious in America that it prevented the growing of sugar beet in areas where heavy attacks had occurred regularly. Infected plants are stunted, the leaves become yellow and curl inwards. The veins of the younger leaves become "cleared" and on the underside of older leaves knob-like swellings are produced. The phloem becomes necrotic, resulting in discoloration of the vascular tissue, and this is apparent as dark concentric rings when the root is cut across. The roots are reduced in size and sugar content, and in the later stages of the disease they develop an abnormal number of hairy, secondary roots.

The virus is not seed-transmitted. It is spread by a leaf hopper (*Eutettix tenellus* Baker) which, once it has become infective by feeding on diseased plants, is capable of causing infections for many weeks. The virus has a wide host range and some of the winter hosts of the vector are commonly infected. The leaf hoppers hibernate on indigenous plants on the foothills of the Rocky Mountains and migrate to beet crops in the valleys during the spring, where they introduce the disease. The numbers of leaf hoppers have been reduced in some areas by destroying the winter host plants and spraying the native vegetation. Losses are reduced by sowing sugar beet early in spring so that the plants make good growth before the crop is infested. Severe losses have been avoided by the use of "resistant" varieties. In infected sugar beet crops it was observed that some individual plants suffered less than others. From such plants varieties have been developed which are more tolerant of infection than the original varieties. The first strains to be developed gave only a moderate yield of sugar, but improved strains have now been obtained which give satisfactory yields in conditions of either light or heavy infection.

Two similar diseases occur in South America. One, Argentine curly top is transmitted by the leaf hopper *Agalliana ensigera* Oman; the other, Brazilian curly top, is spread by the leaf hopper *Agallia albidula* Uhl. The symptoms on sugar beet and host ranges of the viruses differ slightly.

YELLOW WILT

Yellow wilt occurs only in Argentine where its effects have been so severe locally that cultivation of sugar beet has been abandoned. When young

plants are infected they become yellow and stunted, the leaves develop irregular edges, become thick and brittle and curl upwards and inwards. Necrosis develops along the edges and between the large veins. Axillary buds are stimulated and plants have a bunchy appearance. A mass of fibrous rootlets is produced. Such plants usually survive but make negligible growth.

Infection of older plants occurs when temperatures are higher; they wilt during the day and although at first they recover during the night they soon fail to do this and the leaves become scorched. The central leaves are stunted, discoloured and distorted. The roots shrivel from the tip upwards and decay. Plants affected with either yellowing or wilt or with intermediate symptoms occur together in the field. It is spread in the field by a leaf hopper, *Atanus exitiosus* Beamer.

LEAF CURL

Leaf curl, or crinkle, is a virus disease which causes the leaf veins to become translucent, glassy and swollen. Since the veins do not grow as quickly as the interveinal tissue the leaves become puckered. They remain small and are curled inwards, giving the plant the appearance of a cabbage lettuce. The trouble has caused most concern in Central Europe where in some years it has greatly reduced yield; it is not known to occur in Great Britain. It is spread by a lace bug, *Piesma quadrata* Fieb., which hibernates in grass and under trees and hedges around fields. Control is achieved by sowing an early trap crop of beet around the edges of fields and ploughing it in when the bugs have moved on to it before the main sugar beet crop has germinated.

A similar trouble in North America called Savoy is of no economic importance. It is caused by a virus transmitted by *Piesma cinerea*.

NUTRITIONAL DISEASES

NITROGEN DEFICIENCY

Provided there is an adequate supply of moisture, the supply of available nitrogen mainly controls the development of foliage of sugar beet crops. The nitrogen supply in soils is greatly affected by cropping and management, as well as by fertilizer applications. Deficiency is most common on light, gravelly soils in a poor state of cultivation and lacking organic matter. Ploughing in excessive quantities of nitrogen-free organic matter, such as straw, may result in competition for the available nitrogen in the soil, and the following crop will show signs of nitrogen deficiency. Excessive competition from weeds in the early stages of growth of the crop has the same effect. Symptoms resembling nitrogen deficiency may occur in patches when fertility has been reduced by flooding or fire, or as a result of plants being unable to take up the available nutrients because of restricted root development in acid or water-logged soils.

When nitrogen is inadequate the plants remain stunted, the leaves have small leaf blades with long, thin stalks, and the inner leaves are bunched into a small rosette. Older plants are usually prostrate. The leaves are pale green to yellow according to the extent of the deficiency. Dressings of nitrogenous fertilizers lead to recovery: if the dressings are carried to excess, however,

they produce very rank, dark olive green foliage which wilts easily during daytime in summer. The heavy leaf development produced by unbalanced applications of nitrogen gives a misleading impression of the value of the crop, because there is no corresponding increase in root yield; in fact, dressings above the optimum may even cause a reduction in root yield, and will certainly cause a reduction in total sugar because they result in a considerably lower sugar content. They also adversely affect the processing quality of the roots.

In seed crops nitrogen deficiency retards the production of seed-bearing shoots, and results in pale, spindly plants. Unduly heavy dressings of nitrogen produce a spectacular growth on most soils, but are undesirable because they retard the date of harvesting and result in prolonged growth and branching of the flowering stalks which give small, "drossy" seed. Seed crops grown on light soils with excessive nitrogen dressings are liable to wilt suddenly in August if the soil becomes dry, and they ripen prematurely, giving a low yield of small, immature seeds.

PHOSPHATE DEFICIENCY

Acute phosphate deficiency is uncommon in the main beet-growing areas of Britain. It is encountered especially in crops sown after poor grassland has been ploughed up. There is usually difficulty in establishing the crop because the seedlings die from attacks of black leg and fungal infection of the rootlets. Plant losses from these causes continue even after singling. The plants which survive are stunted and usually have dark green foliage. The leaf stalks tend to curl upwards and a blackish-brown necrosis develops from the tips and edges of the leaves without previous chlorosis. Dark brown or black interveinal lesions may develop after wet weather as a result of infection by weakly parasitic fungi. On the ground, attached to the elongated crown, there is usually a whorl of dead leaves. The tap root is stunted and bears a mass of fibrous secondary rootlets (Plate II, upper). Less acute phosphate deficiency is not uncommon after leguminous forage crops, which tend to deplete the soil of phosphates and increase its nitrogen content. Sugar beet plants mildly affected by phosphate deficiency do not show obvious symptoms but if such plants are also affected with virus yellows, they may show an abnormal amount of reddening. The deficiency may be prevented by an adequate dressing of a phosphatic fertilizer. The advice of a soil chemist should be sought if such deficiency is suspected. The normal dressing of superphosphate, 1·0 cwt per acre P_2O_5, is usually adequate even for soils with severe phosphate deficiency.

POTASH DEFICIENCY

The general appearance from a distance of crops affected with potash deficiency is dull, dark olive-green suffused with brown, which suggests the name drab disease. The characteristic necrosis of the foliage which it causes is known as "Scorch" (Plate X, upper and lower). The leaves are thin and soft, and tend to wilt easily. A narrow band of bronze tissue develops around the margins of the middle-aged and older leaves or they may show buff or dull yellow spots which extend inwards between the veins. When this tissue dies it is light or reddish brown and has a tough, papery texture. The necrotic tissue extends deeply in branched lobes between the veins and usually adjoins

green tissue. Although the necrotic areas may curl up and split, they do not easily disintegrate. Bolters are likely to show more pronounced symptoms than normal plants. The symptoms usually develop first in July or August but occasionally appear earlier. They are greatly accentuated by bright, hot weather and drought. The tap root develops less than might be expected from the size of the foliage.

Similar symptoms also occur when sodium is deficient. When diseased leaves are analysed they are often found to be deficient in both elements. Sodium and potassium are both essential for the healthy growth of sugar beet, but so long as a minimum amount of each is available there appears to be some degree of interchangeability of their functions. Consequently symptoms of the disease in the field may be reduced by dressings of either muriate of potash (which supplies potassium) or agricultural salt (which supplies sodium), but usually a dressing of salt is more effective. As with other diseases of this type satisfactory nutrition is largely a matter of balance and when there is a moderate supply of sodium and potassium present in the soil drab disease may be induced by unduly heavy dressings of nitrogenous fertilizers and, to a less extent, by excess phosphatic fertilizers.

If the symptoms of the disease appear before July, some response may be obtained by an immediate top dressing of 2 cwt per acre of agricultural salt, muriate of potash, or of potash salts which contain both sodium and potassium, but it is unlikely that any beneficial results would accrue from later applications. Control lies in preventing the deficiency by adequate and balanced fertilizer applications before sowing. If potash deficiency is suspected a soil analysis will indicate the most suitable dressing, whilst an application of 3–5 cwt per acre of agricultural salt will ensure an adequate supply of sodium for sugar beet crops.

Drab disease occurs most frequently in fields of light sand and peaty fen, or where poor grassland has been ploughed up. It does not occur on strong land. The distribution of the disease is usually associated with physical characters of the field. If there are severe symptoms on some plants, the rest of the crop will usually show slight signs of it, generally by a dull and dark green colour. As the behaviour of potassium and sodium in the soil is similar, the incidence of these two elements tends to be related and both the sodium and potassium content of the leaves of diseased crops is low. The natural incidence of potassium and sodium in soils is disturbed by the application of fertilizers containing these elements. Potassium is usually added in chemical fertilizers, but sodium has not been so freely applied during recent years, especially since sulphate of ammonia has been used instead of nitrate of soda as the main source of nitrogen. During World War II, sodium was more freely supplied to sugar beet in dressings of agricultural salt. In spite of being cheap and giving a good yield response, sodium, applied as either salt or kainite, is now used on only 25 per cent of the sugar beet acreage.

Both sodium and potassium are removed from the soil, not only in the drainage water by leaching, but also in the leaves of crops. Land from which hay crops have been taken continuously without manure or fertilizer additions are seriously deficient. More conservative practices such as folding sheep on beet tops or ploughing in the tops, help to maintain the potassium and sodium content of the soil. The urine of animals contains a large proportion of the potassium consumed in food and it is returned to the land absorbed in straw as farmyard manure.

MAGNESIUM DEFICIENCY

Magnesium deficiency is a common cause of yellowing in sugar beet crops. It occurs every year, but in years when virus yellows is common it is usually obscured. The two diseases are easily confused, for the symptoms closely resemble each other (Plate VII, upper and lower).

The first signs of magnesium deficiency usually appear in July or August, the margins of the middle-aged leaves turning yellow, though in severe attacks the symptoms may appear earlier. At first, only the middle-aged leaves show symptoms, but after a time all leaves except the youngest may be affected, and in autumn even these may develop a marginal necrosis. The yellowing spreads from the margins of the leaves as lobes between the veins, and the line of demarcation between yellow and green tissue is sharply defined. The yellow tissue, which is slightly raised, giving the leaf a blistered appearance, is typically lemon yellow but may assume a deeper shade, and is somewhat thickened so that it breaks when folded. These symptoms are similar to those produced by virus yellows but the colour is paler yellow and the tissues are not so thick or so brittle in magnesium deficiency. Isolated patches of yellow tissue may also develop between the veins in addition to the continuous lobes.

The yellow parts soon become necrotic, beginning at the edges of the leaf where the yellowing starts. Isolated necrotic lesions which somewhat resemble leaf spot diseases develop on the yellow tissue and sometimes closely follow the margin between green and yellow tissue. On other leaves, particularly late in the season, necrotic lesions may develop without any obvious preliminary yellowing (Plate III, lower right). The necrosis extends in lobes between the veins in the same manner as the chlorosis, but it sometimes involves the whole of the distal part of the leaf blade, resulting in a truncated leaf.

The necrotic tissue is dark, purplish brown or nearly black. It tends to curl up and give the margins of the leaves a frizzled appearance, and as it is brittle and disintegrates, the edges of the leaves become very ragged (Plate III, lower left). In humid weather various weakly parasitic fungi, such as *Alternaria*, produce spores on the necrotic tissue, and the isolated necrotic lesions sometimes associated with the disease are also due to infection of the moribund or weakened tissue by such organisms.

In fields where magnesium deficiency is severe, some plants can usually be found which show a uniform yellow chlorosis of the entire leaf. On such leaves, small, brown, necrotic lesions develop freely and the symptoms then closely resemble those observed occasionally in virus yellows. Magnesium deficiency may occur on any type of soil, but is uncommon on fens and silts and most frequent on light sands and gravels. Symptoms of magnesium deficiency are often very prominent on recently limed land. It may occur in varying intensity over the whole field or it may be associated with certain physical features such as drains and plough furrows. It does not occur when farmyard manure has been freely used. Dressings of agricultural salt and potash greatly increase the incidence of the symptoms. Up to the present the disease has not been satisfactorily prevented or cured by soil or spray treatment with magnesium sulphate during or immediately before growing the crop. The solution appears to be to avoid excessive dressings of sodium and potassium and adding magnesium to the soil regularly in the form of

kainite, magnesium limestone, farmyard manure and other magnesium containing fertilizers. Symptoms of the disease are more intense on some varieties than on others, but as little is known of the extent to which the disease affects yield, the selection of varieties solely on their apparent tolerance of this deficiency has not been justified.

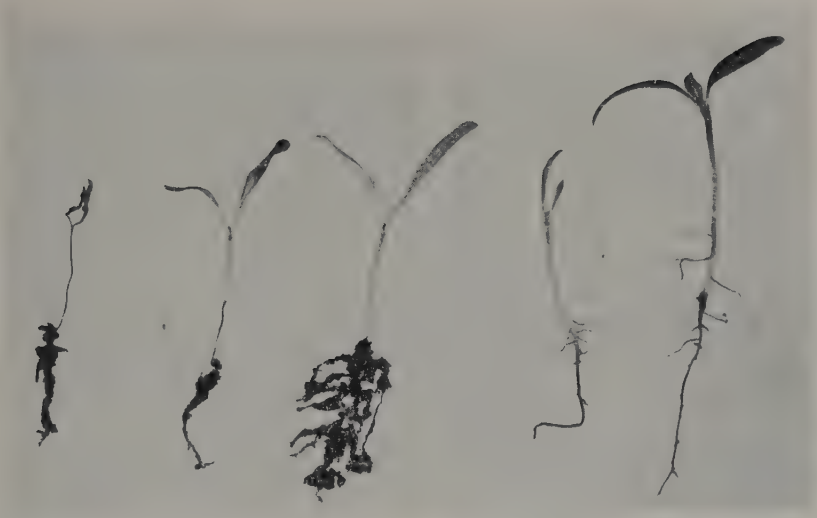
MANGANESE DEFICIENCY

Speckled yellows, the name given to a disease caused by manganese deficiency, may develop on sugar beet plants at any time of the year. In the root crop it usually appears shortly after singling. The intensity of the symptoms fluctuates during May, June and July, but in August it decreases appreciably, and by October the plants often appear to have made a complete recovery.

Badly affected fields can be recognized from a distance by the pale greenish-yellow colour, and by the stiff upright habit of the plants (Plate IX, upper). This upright habit is due to the petioles of the outer leaves growing almost vertically, whilst the margins of the leaves roll inwards, thus giving the leaves a narrow, pointed appearance. Less severely affected plants are of more normal habit, but still show the characteristic yellow, interveinal mottle of the foliage. If the symptoms are slight the pattern is of lobed, yellow inter-veinal patches which produce a starry mottled effect, but if the symptoms are severe, the yellow patches fuse and the green tissues along the veins stand out like a fern-leaf pattern which is particularly obvious by transmitted light. The chlorotic tissue is pale yellow to greenish-white. Small, angular, and sunken necrotic patches which are pinkish-buff, translucent, and of a horny texture, develop in the centres of the chlorotic patches. The necrotic tissue often splits and falls out, so that later in the season when the plants have recovered somewhat, the leaves show numerous holes surrounded by healthy green tissue. If the necrosis has not reached such an advanced stage when the plants recover, the leaves are left with brown, angular, depressed, necrotic spots.

Although there is adequate manganese in most soils, its availability to the plant is critically affected by certain soil conditions, of which the most important are acidity and organic matter content. Manganese deficiency symptoms often develop on plants grown in alkaline, peaty soils. Disease symptoms also develop in crops other than sugar beet grown in these soils; for instance, grey speck in oats and marsh spot in peas. Speckled yellows also occurs commonly on sandy soils reclaimed from heath, on old grassland during the first two years after ploughing, and on sewage farms. It may be induced in a severe form in crops taken immediately after excessively heavy liming. The intensity of the symptoms is not appreciably affected by soil moisture, but affected plants suffering also from drought may be excessively stunted. The disease may occur all over a field or in irregular patches corresponding to soil variations. Its presence is often associated with gravelly patches in the subsoil, and its absence with clay patches. When the disease is not severe, it is often found that one plant shows pronounced symptoms whereas neighbouring plants show none; this is probably the result of the varying susceptibility of different plants to manganese deficient conditions.

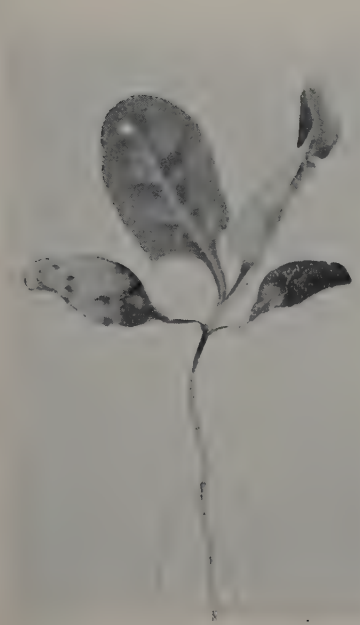
Crops affected with the disease may be cured in a most spectacular manner by applications of manganese sulphate. The disease may be cured later by applying 20–60 lb per acre of manganese sulphate to the plants,



(*a*) (*b*) (*c*) (*d*) (*e*)

Seedling Diseases (see p. 1)

(*a*) A seedling killed by black leg (*b*) An earlier stage of black leg (*c*) A healthy seedling (*d*) and (*e*) Acid soil injury to the primary root



Acid soil injury to an older plant

Strangles (see p. 6)

PLATE I

PLATE II

Dwarf (cucumber mosaic virus)
Note the relative size of the leaves and stems of healthy seed plants in the background (see p 18.)

A plant suffering from phosphate deficiency
(see p. 21)

A chimera (see p. 41)

[*Photo: A. R. Wilson*

Tip burn: separated heart leaves (see p. 28)

[*Photo: V. Stansfield*

Magnesium deficiency: the ragged edges of a leaf where the necrotic tissue has disintegrated (see p. 23)

[*Photo: V. Stansfield*

Magnesium deficiency: necrosis developing on a green leaf

PLATE III

PLATE IV

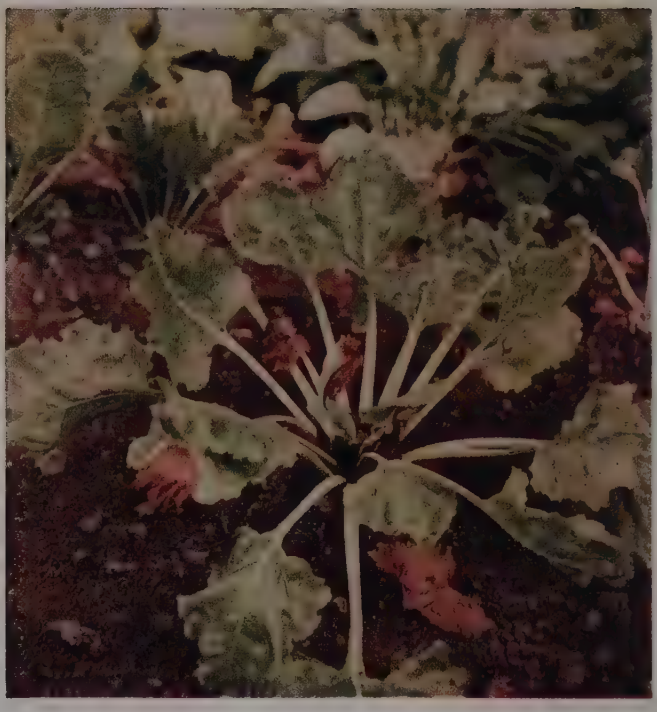
Heart rot (boron deficiency) (see p. 25)

A root affected by heart rot, cut lengthwise

Virus yellows (beet yellows virus): a severely affected plant which was infected early (see p. 10)

A seed plant infected with both virus yellows (beet yellows virus) and Mosaic (beet mosaic virus) (see pp. 11 and 17)

PLATE V



Heart rot (boron deficiency) (see p. 26)

A plant recovering from an attack of downy mildew (*Peronospora schachtii*) (see p. 29)

PLATE VI

Virus yellows (see pp. 10 and 23)

Magnesium deficiency (see p. 23)

PLATE VII

Downy mildew (*Peronospora schachtii*) (see p. 29)

A yellow leaf from a plant attacked by downy mildew (see p. 29)

Speckled yellows (Manganese deficiency) (see p. 24)

Mosaic (beet mosaic virus) (see p. 17)

PLATE IX

Potash deficiency; early chlorotic symptoms (see p. 21)

Potash deficiency: necrotic stage which follows that shown in the illustration above

PLATE X

Cercospora leaf spot (*Cercospora beticola*) (see pp. 34 and 35)

Ramularia leaf spot (*Ramularia beticola*) (see p. 35)

PLATE XI

Rust (*Uromyces betae*) (see p. 33)

Powdery mildew (*Erysiphe* sp.) (see p. 32)

PLATE XII

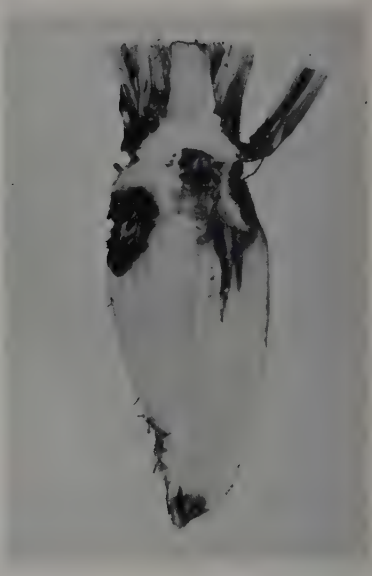
A seed plant infected by downy mildew (*Peronospora schachtii*) (see p. 29)

Dead heart leaves resulting from downy mildew infection (see p. 29)

Downy mildew: a plant which has recovered from early infection and developed an elongated crown (see p. 29)

PLATE XIII

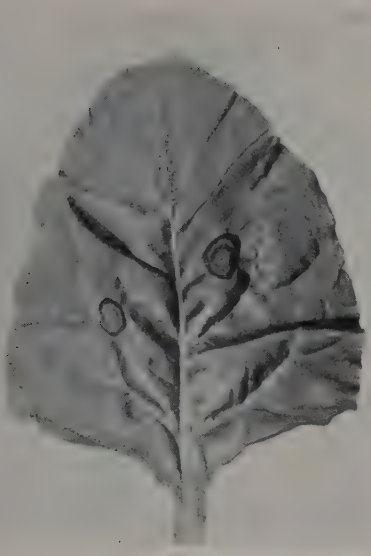
Lightning injury (see p. 38)



[*Photo : A. R. Wilson*
Section of a root struck by lightning

Lightning injury to young plants
The two outer plants have been cut lengthwise to show the internal injury
The centre plant has produced a mass of fresh lateral roots

PLATE XIV



[*Photo : C. E. Foister*

Pleospora leaf spot (*Pleospora betae*)
(see p. 35)

[*Photo : A. R. Wilson*

Stem rot: the stalk of a seed plant showing spore cases of *Pleospora betae* (see p. 46)

Capsid bug injury (see p. 40)

PLATE XV

Violet root rot: mycelium and infection cushions of *Helicobasidium purpureum* on the surface of the root (see p. 41)

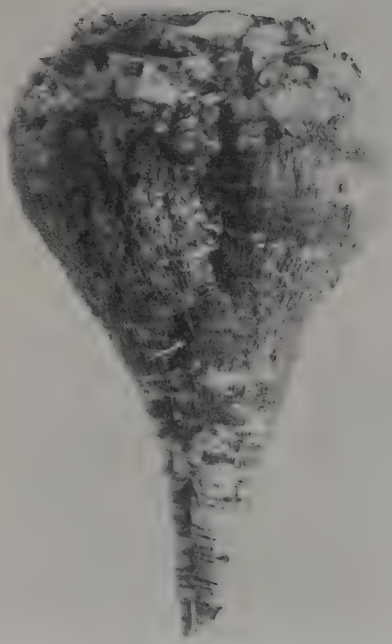
A washed root showing a more advanced stage of violet root rot

Fusarium rot (*Fusarium culmorum*) (see p. 47)

Wet rot (*Phytophthora megasperma*) (see p. 43)

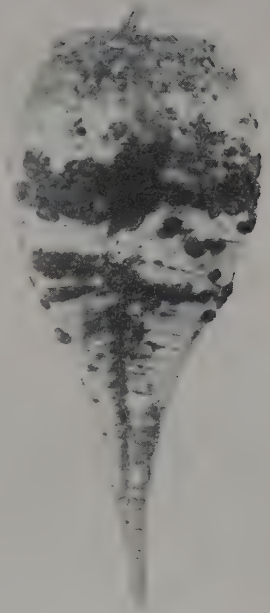
PLATE XVI

PLATE XVII



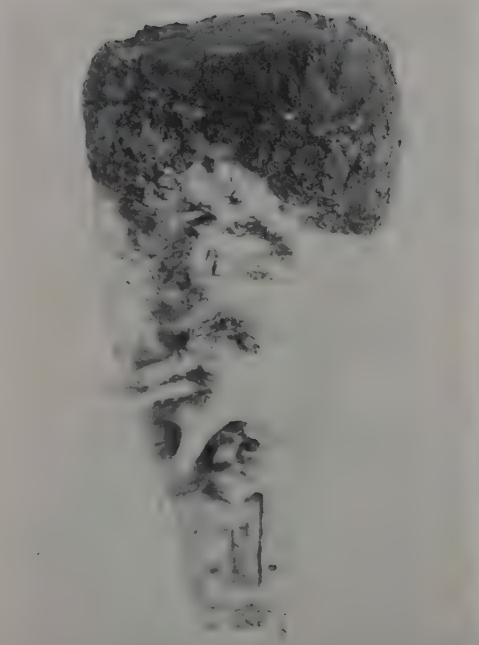
Scurfy Root (see p. 45)

Lenticel Rot (see p. 50)



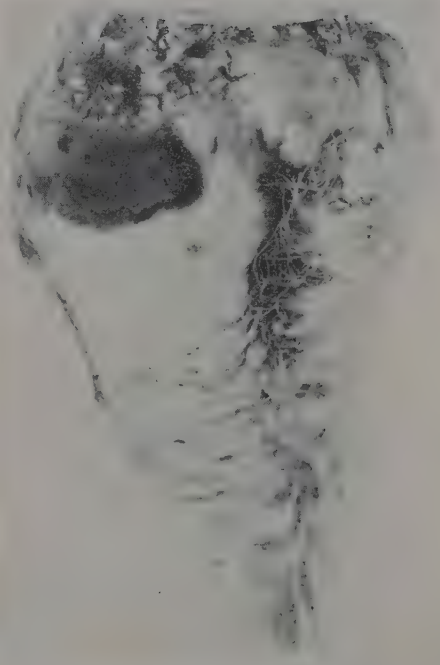
Scab (*Actinomyces* sp.) (see p. 44)

PLATE XVIII



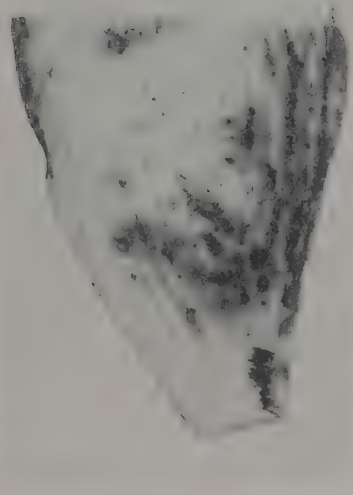
Eelworm Canker (*Anguillulina dipsaci*) (see p. 51)

Section of a root with severe Eelworm Canker

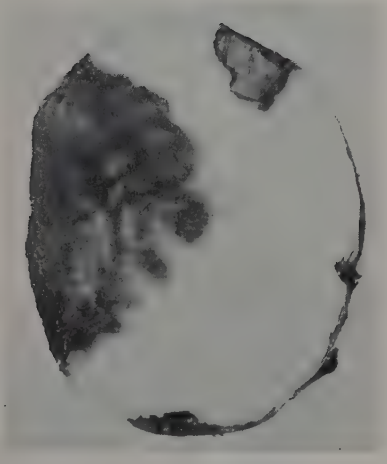


Crown Rot (*Phoma betae*) (see p. 45)

[*Photo : H. H. Glasscock*
A mangold with Black Wood Vessel disease (see p. 36)



A sugar beet affected with Internal Fleck (see p. 48)

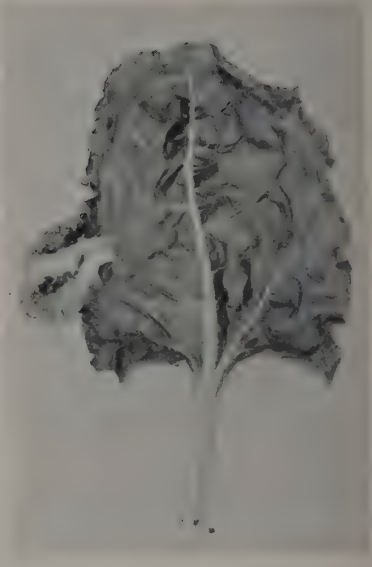


[*Photo : A. R. Wilson*
Section of a root with Clamp Rot (*Botrytis cinerea*) (see p. 47)

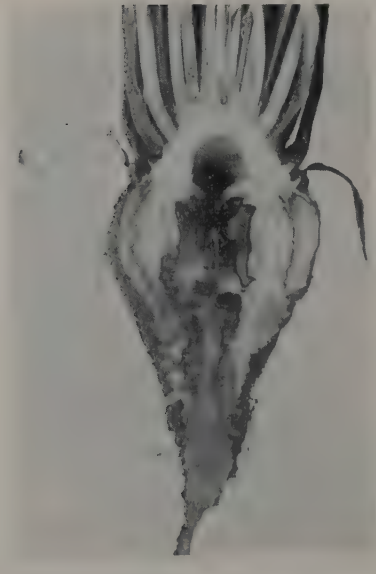
[*Photo : A. R. Wilson*
Section of a frozen sugar beet from which bacterial slime is oozing (see p. 49)

PLATE XIX

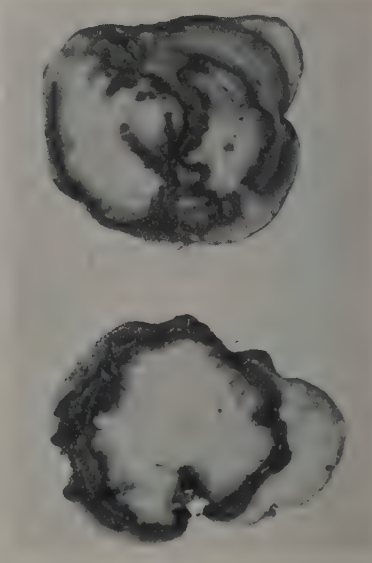
[*Photo: A. R. Wilson*
Crown Gall (*Agrobacterium tumefaciens*)
(see p. 45)



Crown Gall (*Agrobacterium tumefaciens*)
on a sugar beet leaf (see p. 45)



Section of a root affected with Internal
Zoned Rot (see p. 50)



Sections of roots affected with Internal
Zoned Rot (see p. 50)

PLATE XX

either by dusting the foliage when the plants are wet with rain or dew, or by spraying it on in solution. It is desirable to treat affected plants in May or June, as soon as the symptoms are noticed, but the practical difficulty on a farm scale is to get the dust or spray accurately applied to the plants, for there is usually little foliage to receive the spray or dust at that time. Early treatments may need repeating after three or four weeks. After treatment the plants become green in ten to fourteen days.

As affected crops often make a natural recovery during the summer it is difficult to decide whether treatment is worth while. In some experiments, a response in yield of about 1 ton per acre of roots and up to 0·5 per cent in sugar content has been obtained, but in others, although the treatment has reduced the symptoms on the leaves in June and July, it gave no measurable response in yield when the plots were lifted in October. When the disease is severe there is no doubt that treatment is worth while if it is applied in time, because in addition to the increased yield, the more vigorous growth of the plants gives a better cover and helps to keep down weeds, which are usually troublesome in severely affected crops.

In fields where the soil is naturally conducive to the trouble, the best policy to follow is one of soil amelioration rather than of sporadic control. Applications of manganese sulphate to the soil are uneconomical, as the manganese is soon rendered unavailable to the plant whilst the soil remains alkaline. Acidification of the soil by an application of 1 ton per acre of sulphur has a more lasting effect, and the use of acidifying fertilizers such as sulphate of ammonia is recommended. Heavy dressings of farmyard manure result in a reduction of symptoms of manganese deficiency on the crop. It is claimed that dressings of basic slag, which has a considerable manganese content, have led to an increase in the uptake of manganese in oats, but a similar effect has not been proved in sugar beet. Nevertheless, instances have occurred where a heavy dressing (12 cwt per acre) of a compound fertilizer has given a better control of manganese deficiency symptoms and a greater increase in yield than applications of 1 cwt per acre of manganese sulphate.

MANGANESE TOXICITY

The toxic effects resulting from the uptake of excessive quantities of manganese are frequently seen in Great Britain. Affected plants are severely stunted and the foliage is usually a uniformly pale yellowish-green without any mottling or speckling; occasionally a mottle may develop later in the season. Affected plants are upright, the margins of the leaves tend to roll, and the outer leaves wither prematurely. This trouble is associated with acid soils, generally with a *p*H below 5. It usually occurs in well defined patches, the centrally placed plants being the worst affected, but it may also be irregularly dispersed over a part or the whole of a field. It does not necessarily occur on all acid soils.

BORON DEFICIENCY

Boron deficiency causes heart rot and dry rot, the first name being applied to the symptoms on the above-ground parts and the second to those on the

tap-root. The disease is of considerable economic importance. It is also interesting because the cause was at first the subject of much speculation and was confused with secondary, accentuating factors. Later, it was proved that a little boron is essential for the healthy development of sugar beet, and if the crop does not get enough, either because of the absence of the element from the soil or because soil conditions have rendered it unavailable to the plant, the disease develops.

The first sign of the disease, which gives rise to the name heart rot, is the death of the growing point and the young leaves (Plate VI, upper). Affected plants are prostrate because the young leaves are stunted and the petioles of the older leaves are more or less horizontal. The surface of the groove on the upper side of the leafstalk, particularly near the base, is often scarred with corky brown tissue and transverse cracks. The blades of the older leaves are thickened and silvery, often bearing irregular scar-lines along what appear to be cracks in the surface. In the early stages, when the youngest leaves have wilted and the growing point has turned black and necrotic, the next older leaves often show an interveinal yellowing. This is because they have been partly cut off from the root by the death of the tissues at the bases of the petioles. Later, there is considerable yellowing and necrosis of the old leaves also and the plants may even become almost defoliated. A severely affected crop has a very drab, wilting appearance. With the advent of more favourable growing conditions, however, affected plants may develop fresh leaves from lateral growing points.

The disease also results in decay of the tissues around the shoulder of the tap root (Plate IV, lower). At first there is a superficial, greyish-brown discoloration of the root tissues extending downwards from the leaf bases, or localised lower down at the widest part of the root, apparently quite dissociated from the symptoms on the overground parts. When the root is cut through, streaks of grey, discoloured tissue are seen between the vascular rings (Plate IV, upper). At first only the tissue near the surface is discoloured but later the deeper tissues may be involved. The affected tissues decay, usually as a result of secondary attacks by *Pleospora betae* or by species of *Fusarium* (most frequently *F. culmorum*). If *Pleospora* is present its small black spore cases (pycnidia) may be found on the surface of the decayed tissues. If the tissues are invaded by *Fusarium culmorum* any cavities in the crown, or even the exterior in humid conditions, may be covered with a fluffy white mycelium which is diffused in places with red or gold, and beneath which, at a later stage, slimy golden spore masses appear. The rot may envelop the whole of the crown, in which case the plant is soon killed. In other plants, less severely affected, fresh leaf growth is produced and the rot remains superficial. The affected tissue sloughs away leaving a sunken, corky lesion around the crown. This has a yellowish-brown surface and at the margins there is a raised edge of healthy tissue which sharply defines the lesion. The rot tends to be most active in the discoloured, intervascular tissues, and the vascular rings persist intact longer with the result that they project at the margins of the lesion. The tissues immediately below the growing point are also discoloured and decayed, and this condition may extend as far as the pith cavity in the centre of the crown of the root.

The disease is uncommon in seed crops, because they are not usually grown on soils where it is likely to occur, but occasionally isolated affected plants have been observed. The "bolters" which occur in root crops grown

on fields subject to the disease often show pronounced symptoms. The main flowering stem is stunted and the growing point dies. Other stems may shoot from the crown and grow longer than the original, but eventually their growing points also die. The growing points of laterals along these stems also give rise to stunted growths which appear as small rosettes of discoloured bracts, and eventually these growing points also die.

Heart rot occurs sporadically on some soils while on others it is never seen. It is most common on alkaline soils. Liming increases the likelihood of the disease and if carried out to excess may result in the appearance of the disease on soils where previously it had never occurred. Heart rot is associated with soils of low organic matter content, and consequently heavy dressings of farmyard manure tend to reduce it. It is particularly prevalent in dry summers. Early sown crops usually develop more severe attacks than late sown crops, and crops in which vigorous growth has occurred early in the summer are particularly liable to be affected if there is a late summer drought. The disease may be increased by heavy dressings of nitrogenous fertilizer, and also by applications of salt. Such treatments encourage vigorous growth and it is probable that the available boron is all used and fixed in the older leaves, so that the growing points are eventually starved of their essential requirements.

The disease usually occurs in irregular patches in fields and the losses are difficult to assess. Control may result in a considerable increase in the yield of total sugar. In an experiment carried out in Norfolk this increase was assessed at 35 per cent: this was partly due to an increase in the root yield and partly to an increase in the sugar content of the roots which is usually very low in crops affected by heart rot. The disease also increases the noxious nitrogen content of the root juices and consequently the processing quality of the roots is impaired. Apart from the reduction of sugar there are other disadvantages; the yield of tops is lower, harvesting more difficult, and the harvested roots are particularly liable to rot when stored in clamps for any length of time.

The disease is controlled by applications of about 20 lb per acre of borax. If borax is applied in July when symptoms of the deficiency are first seen, the development of the trouble will be arrested, but it is usually too late for the full benefit of treatment to be obtained. The disease may be prevented by applying borax to the seedbed. In districts where the disease is known to occur, especially where special efforts are being made to obtain good crops by liming, early sowing, and heavy applications of fertilizers and salt, it would be a wise precaution to apply also a dressing of borax. Complete control of the disease has been achieved in areas where previously it was very severe, by an initial application of 30 lb of borax per acre followed by 20 lb per acre to each subsequent sugar beet crop as it occurred in a four course rotation. Boron compounds are easily leached from the soil so that there is no danger of toxic concentrations of boron being reached with such applications.

Borax may be applied mixed with sand or incorporated in the fertilizers. It should not be mixed directly with sulphate of ammonia, but may be incorporated in a compound after being mixed with superphosphate. There is a certain amount of boron in natural nitrate of soda, but the amount is not enough for therapeutic purposes. The results of many experiments have shown that there is no response in yield of sugar beet to borax applications

to crops which show no signs of boron deficiency, so that the general incorporation of borax in all compound fertilizers, except for use in those areas where there is likelihood of the disease occurring, is not recommended.

CALCIUM DEFICIENCY

A disease called tip burn occurs late in the season on a small proportion of plants in some fields, usually on light sandy soils. It is attributed to an inadequate uptake of calcium by the plant, but is not necessarily associated with an acid soil which needs liming. Although the disease can be produced in pot cultures by omitting calcium from the nutrients, the factors concerned with the development of the disease in the field are much more complex. It may occur in fields where the calcium content of the soil is high, probably because the plants are unable to absorb enough of this element in comparison with the supply of other nutrients. Like many other nutritional diseases it is the outcome, not of an absolute deficiency of the particular element, but of an unbalanced nutrition which results in a relative deficiency. Tip burn is often severe in sugar beet crops on land which has been flooded by sea water.

The outer leaves of calcium-deficient plants appear normal, but the inner ones consist merely of leaf stalks with minute leaf blades (Plate III, upper right). A black necrosis develops at the tips which curl inwards. In extreme cases the youngest leaves are so severely affected that the disease may resemble heart rot. In less severe cases the margins of the inner leaves are fringed with a narrow band of dark brown or black necrotic tissue. This retards growth of the margins whilst the centre of the lamina grows normally, resulting in the leaf becoming spoon shaped. The leaf blade is buckled and the tension may cause the dead edges to split. The disease is not of commercial importance.

IRON DEFICIENCY

Iron deficiency is rare in sugar beet crops in Great Britain. It causes an interveinal yellowing, which in severe cases may result in a network of green veins on a yellow background. More often there is only a slight, transient mottle, particularly on the younger leaves, which is difficult to distinguish from speckled yellows and mosaic. It is associated with alkaline soils and a high phosphorus content, both of which render iron unavailable to the plant.

OTHER DEFICIENCIES

Diseases caused by a deficiency of other elements have been reported in sugar beet crops in various parts of the world, but they are not known to occur in Britain. Zinc deficiency produces irregularly shaped grey-brown or slate coloured necrotic spots surrounded by a margin of light green tissue. Eventually the whole leaf becomes necrotic and curls upwards. Plants suffering from Copper deficiency have a dark, bluish-green appearance. The outer leaves become chlorotic, first at the tips, and later over the whole leaf except the veins, which gives them a mottled appearance. The necrosis which follows is greyish-brown or greyish-white.

MILDEWS

DOWNY MILDEW

Downy mildew, which is caused by the fungus *Peronospora schachtii* Fuckel, has increased in importance in recent years in England and in some seasons reaches serious proportions in both seed and root crops. The development of severe outbreaks depends upon infection persisting in an annual cycle between root and seed crops, and the worst attacks therefore occur in those parts of the Midlands and East Anglia where the two crops are grown together. The disease is only sporadic in other parts of the country, and in some parts, e.g., north Yorkshire and Scotland, is rare.

Symptoms

The disease shows definite stages of development and decline. The fungus grows within the overground parts of the plant, but does not kill the tissues immediately. Infected tissues become thickened, brittle, puckered and distorted and are usually light green (Plate VIII, upper). The fungus generally infects the growing point and the rosette of young leaves, but in conditions favourable for infection, lesions may be produced on older leaves. It is not uncommon to find leaves in which the lower parts of the leaf blade are infected and the distal parts healthy. A downy growth covered with spores develops on the surface of infected leaves, and this gives them a purplish hue, later changing to buff-grey. Spore production is favoured by mild, humid conditions and is normally more luxuriant on the undersurface of the leaf.

Young root crops are occasionally attacked while still in braird, but usually the disease first becomes apparent on plants after singling in May or June. The rosette of infected leaves eventually dies, becoming brown and dry in fine weather, or rotting if it is wet. The decay may spread throughout the crown and kill the plant, but generally the plant makes fresh growth by developing other growing points or masses of small leaves from lateral buds (Plate VI, lower). In the meantime fresh infections occur on other plants and throughout the season fields contain plants in all stages of infection and recovery.

In the later stages of downy mildew attack, the older leaves, which were at first unaffected, become yellow, thickened and brittle as a result of the damage caused by the fungus in the region of the growing point. This may be so extensive as to give the whole field a yellowish appearance. The yellowing is very similar to that caused by beet yellows virus, and it is often impossible to decide in the field whether individual plants are suffering from the effects of one or the other, or both diseases. Chlorosis (yellowing) resulting from downy mildew may sometimes be distinguished by obvious signs of infection on other parts of the plant, or by the discoloration of the petiole, necrosis of the main vein and exudation of sugary drops on the underside of the older leaves and petioles, which are characteristic of the disease (Plate VIII, lower).

When the heart leaves are dead it is often difficult to decide whether a plant has been affected with downy mildew or heart rot (p. 25). However, a young leaf or portion of a leaf that has not become necrotic may show the characteristic thickening and colour of tissue infected with downy mildew (Plate XIII, lower left), or the petioles of the outer leaves may have the

necrosis and discoloration which characteristically follow downy mildew injury to the growing point. If the plant is suffering from heart rot, the outer zones of the rot will be discoloured or there will be external lesions extending downwards from the crown.

Effect on Yield

The effect of downy mildew on yield of roots has been roughly determined by marking plants as they become infected, and weighing them after lifting. These experiments have indicated that plants infected before the end of July weigh only half as much as healthy plants and the loss decreases with later infections. It is characteristic for the plants which are infected towards the end of the season to be heavier than uninfected ones and this suggests that the heavier yielding types are more susceptible to the disease. Early infection causes a reduction in sugar content of 1–2 per cent, and a considerable reduction in purity of the root juices (a measure of the extent to which sugar can be extracted from the roots in the factories).

The ill effects of the disease also extend to less easily measured difficulties in harvesting. The leaf stalks of infected plants are very brittle so that they break easily when gripped for pulling and such plants are often left in the ground. Furthermore, when plants make fresh growth after recovery from infection, the crown becomes misshapen (Plate XIII, lower left) and may split. Such crowns are undesirable for they result in excessive top tare, and although decay may not progress very far whilst they are still growing, it often continues after the roots have been harvested and clamped.

In Seed Crops

In steckling beds, infected plants may be found within three or four weeks of sowing. The symptoms at first resemble those on root crop plants (Plate XIII, upper) but often seed plants are killed; others less severely attacked send up attenuated seed-bearing stalks on which infected laterals are closely packed. Often such plants die, but under favourable growing conditions one or two of the lateral branches may develop normally, though the yield of seed from such plants is negligible. Later infection developing on seed plants which have already produced seed bearing branches is normally confined to young lateral buds, but sometimes the fungus attacks the developing seed clusters. The stand of plants may be seriously reduced by downy mildew.

Life Cycle of the Disease

The downy mildew fungus may form resting spores (oospores) in diseased tissues, and they may sometimes be found in the seed. Seed-borne infection is not thought to be important in this country, because in many areas where the climate would favour the development of the disease, crops remain uninfected. The summer spores (conidia) which are produced in large numbers on the surface of infected leaves are disseminated through the air and are the most important means by which the disease is spread. They will germinate only in humid conditions and outbreaks of the disease can often be associated with spells of damp weather about two to three weeks previously. Spread of infection is restricted by hot, dry weather. Although spores are not produced freely at low tempretaures, they can still germinate and produce infection. Thus, provided there is a source of spores, the long wet periods experienced during the autumn and winter favour spread of the disease in steckling beds.

Ground-keeping beets found in corn and other crops following the previous year's sugar beet crop are sometimes infected with downy mildew, and infection may spread from them to nearby root crops. This infection survives from the previous year because the fungus is able to lie dormant in the crown of the root and develop in and on the new leaves the following year. Similarly, the fungus may survive over winter on closely related susceptible plants such as mangolds, sea-kale beet and garden beet. Wild beet (*Beta maritima*) is very susceptible and may form a source of infection for sugar beet crops in coastal districts. The downy mildew occurring on goosefoot (*Chenopodium album*) closely resembles the one on sugar beet, but will not infect it: nor will the beet downy mildew attack goosefoot.

When seed crops and root crops are grown in close proximity there is a continuous sequence of plants throughout the year on which the disease can thrive. The stecklings, sown in June or July, become infected by spores blown from infected root crop plants. The disease spreads in the steckling bed throughout the autumn and winter. Some of the plants set out to produce seed are usually infected and produce large numbers of spores in May and June which spread to nearby young root crops, where infection continues throughout the summer, according to the weather conditions.

Very heavy infection is normally experienced in root crops grown adjacent to seed crops. The severity of the attack varies according to the size of the seed crop and the incidence of downy mildew in it. At first the heaviest infection is in the immediate vicinity of the seed crop, but later in the season the gradient is less pronounced as a result of secondary spread within the root crop. Although severe infection from an individual seed crop may be localised, occasional spores may travel to far greater distances, and so start centres of infection. Indeed, in a seed growing area, the atmosphere becomes charged with spores, and no fields escape the possibility of infection.

Control

Control of the disease in the root crop is not easy. Varieties differ very considerably in their susceptibility to attack, but as this feature does not appear to be constant from year to year it is not possible to give a list of varieties in their order of susceptibility. Spraying root crops with fungicides is not a practical or effective method of control, especially when there is a nearby source of infection such as a seed crop. The best control measure is to break the cycle of infection from root to seed and seed to root crops. This can be achieved by planting only healthy stecklings, and the steckling bed is the obvious place in which to carry out control measures for it covers an area of approximately only one-tenth of that of the seed crop planted from it.

Whereas stecklings raised in beet or seed growing districts are almost invariably exposed to infection, it is possible to sow stecklings in districts well isolated from root and seed crops. A similar measure of isolation is recommended for avoiding infection with virus yellows. Nevertheless, even very light initial infection may spread within the steckling bed if conditions are favourable. For this reason it is advisable to choose exposed, well drained sites, because the damp conditions persisting in low-lying, sheltered situations will favour spread. Similarly, fields at a high altitude where dew persists and fogs are frequent favour the disease.

Some control may be obtained by spraying the steckling beds in autumn with a fungicide. Bordeaux mixture, proprietary copper fungicides, or zineb

are used; they are usually incorporated with the insecticide spray used for controlling virus yellows. As seedlings are most susceptible to infection when they are young it is essential to make the first application as soon as leaf development begins. Further applications should be made at fortnightly intervals with the object of keeping the foliage, and especially fresh growth, covered with spray. Sprayed plants are rather susceptible to frost damage, and it is not advisable to continue spraying after November.

Either early sown (July) or late sown (August) stecklings may give the more heavily infected seed crop according to the season. The important factor is the weather at the time when the seedlings are very young and highly susceptible. If the weather is bright, dry and free from dews following the early sowing, and humid following the late sowing, the August sown plants will become more heavily infected. On the other hand, early sown plants are exposed to infection for longer than late sown plants, so that the chances of infection spreading among them are increased. Furthermore, if spraying is carried out, a greater number of applications will be required for early sown than for late sown stecklings.

Stecklings infected with downy mildew are readily killed by frost: consequently the proportion of infected plants in the bed is considerably reduced after a hard winter, and seed crops are then comparatively free from infection. Conversely, after mild winters severe attacks are experienced, not only because diseased plants survive, but also because the disease continues to spread in the steckling beds during mild winter weather. If, however, the stecklings are transplanted in the seed field during autumn or early winter the disease is likely to spread less among the widely spaced plants than among the closely packed plants in a steckling bed.

Plants obviously infected at transplanting time should be discarded and destroyed. However thoroughly this is done there will always be some which are actually infected but do not show symptoms until conditions are more favourable for the development of the disease in April and May. The seed crop should then be inspected again and all infected plants removed by cutting them off with a spade and burying them on the spot. If they are pulled up and carried about the field, there is a great risk of distributing spores over the crop.

POWDERY MILDEW

A powdery mildew* sometimes develops on the foliage of sugar beet. It is favoured by hot, dry weather and consequently occurs most freely in eastern and southern England. In hot, dry years the disease may be found in August, and by September it may have affected many plants.

The mildew grows on the surface of the leaf as a cobweb-like film of white mycelium (Plate XII, lower). Chains of spores are borne in profusion on this mycelium and the fungus is spread by their dispersal through the air. Infected leaves at first retain their green colour underneath the mycelial weft, but later, as the fungus penetrates and draws nourishment from the underlying cells, the leaves turn pale yellow and eventually become brown, dry

* The identity of this mildew is still uncertain. In Russia and America it has been named *Erysiphe polygoni* DC. and in Europe it is usually called *Microsphaera betae* Vañha. The perfect stage has been seen only once in England and then it was not fully developed, but it appeared to be a species of *Erysiphe*.

and shrivelled to a papery texture. In favourable conditions the fungus spreads rapidly over all the leaves, giving the plant the appearance of having been dusted with a fine, greyish-white powder. The older leaves killed by the fungus collapse on the ground around the plant, but remain attached to the crown: the weft of white mycelium is still apparent on them.

LEAF SPOTS

RUST

The beet rust fungus, *Uromyces betae* (Pers.) Lév., produces brown or reddish spots about the size of a pin-head on both the upper and lower surfaces of the leaves (Plate XII, upper). The lesions are either scattered over the leaf blade and leaf stalk, or may be grouped in rings about $\frac{1}{8}$–$\frac{1}{4}$ in. in diameter. There is often no discoloration around the pustules, but they may be surrounded by a yellow or reddish-brown halo. Leaves showing symptoms of other diseases such as virus yellows are often also attacked by rust. The pustules may be dispersed or may be so densely aggregated that the leaf becomes brown and necrotic in patches and withers. During summer and autumn spores (uredospores) are produced in the pustules. They are normally dispersed in the air but are sometimes so numerous that they appear as a russet powder which is washed down the groove of the petiole and dusted over the surface of the leaf, accumulating in crevices and depressions. In autumn the pustules become brown because darker coloured winter spores (teleutospores) are produced. These spores survive on pieces of dead leaf tissue and germinate in the spring to produce a third type of spore (sporidium) which infects the young foliage. From these infections clustercups develop in April and May on the older leaves and petioles of stecklings; they also occur on the shoots which develop on clamped mangolds. They are bright orange-yellow and occur in groups which produce raised blisters and distort the leaf or leaf-stalk. Yellow spores (aecidiospores) are produced in them and these re-infect the leaves.

The fungus survives the winter mainly in seed crop stecklings, clamped mangolds, groundkeeping beets and mangolds, and wild beet. It spreads from these sources to young sugar beet crops in spring and early summer. The foliage of beet and mangold seed crops is sometimes heavily infected and severe attacks in root crops can arise from this source. The fungus is widely disseminated by airborne spores. The rate of spread of the disease is affected by atmospheric conditions, and rust attacks are often severe in crops on fenland and in low-lying areas where the leaves tend to remain wet for considerable periods. Spores may be carried on the seed coat and instances have been recorded abroad in which the disease was believed to have started in this way.

Occasional plants are often more severely infected than their neighbours, probably because plants of different genetical constitution vary considerably in susceptibility to the disease. It should be possible to select strains of sugar beet resistant to rust, but the damage so far caused has not justified the attempt. However, some commercial varieties are reported to be more susceptible than others. Whatever the variety the rank foliage produced by unbalanced and excessively heavy dressings of nitrogenous fertilizer is susceptible to rust infection.

CERCOSPORA LEAF SPOT

Cercospora leaf spot is favoured by hot climates and is one of the major problems of beet cultivation in central and southern Europe and certain parts of north America. In Great Britain severe attacks occur only occasionally in the south and south-west; elsewhere infection is usually confined to small patches or occasional plants. The disease is caused by the fungus *Cercospora beticola* Sacc. and is spread by airborne spores produced on the leaf lesions. The fungus thrives at high temperatures, but does not become epidemic unless high temperatures (20° C or more) are combined with high humidity. Plants infected with virus yellows are much more susceptible to *Cercospora* than healthy ones.

Circular lesions are formed on the leaves, 3–5 mm in diameter, grey and necrotic in the centre with a reddish-brown margin (Plate XI, upper). The spots are isolated at first but in severe attacks they coalesce and the leaf shrivels. Consequently, badly attacked plants are surrounded by a ring of dead, brown leaves, still attached to the crown but lying on the ground; the grey spots, encircled by a dark ring, can still be distinguished. The youngest leaves are seldom affected. If the spots remain discrete on otherwise sound leaves the necrotic centres may drop out, giving a shot-hole appearance. The destruction of the foliage severely reduces yield and produces a low sugar content and low purity of the root juices.

The fungus can persist on dead leaves and in the soil for a considerable time. Wide rotation of crops of the beet family is therefore an essential control practice in areas where the disease becomes epidemic. Carting diseased tops from the field to feed directly to cattle or for ensilage helps to reduce the amount of infective material in the soil. The fungus is also able to infect a number of common weeds which, in addition to increasing the humidity amongst the beet plants, also act as carriers of infection.

The disease is seed-borne and it is doubtful whether dry treatment of seed with organo-mercury dusts provides an effective control. Steeping the seed in wet fungicides is said to reduce initial attacks arising from seed-borne infection. Apart from general sanitary measures, spraying crops periodically with 4–4–50 Bordeaux mixture is resorted to in some countries to avoid epidemics. The first application is made before the disease normally appears (June) and this is followed by four or five further applications at fortnightly intervals. In countries where it is usually too dry to favour the development of epidemics, close spacing of the plants, which causes a higher local humidity among the leaves, may lead to outbreak. Experiments have shown that in these circumstances greater yields are obtained from wide spacing than from close spacing.

Varieties of sugar beet have now been developed which, it is claimed, are resistant to the disease and yield much better than susceptible varieties in conditions favourable to infection. Up to the present, the disease has not caused sufficient damage in this country to warrant the use of special control measures.

RAMULARIA LEAF SPOT

A leaf spot caused by *Ramularia beticola* Fautr. & Lamb. occurs occasionally in Great Britain, but rarely causes appreciable damage. The necrotic lesions

are circular, about ¼ in. in diameter, and grey or light fawn (Plate X, lower). They are usually larger than those caused by *Cercospora* and may or may not be encircled by darker brown tissue. When the lesions are numerous they fuse together and destroy the leaf. On the surface of the leaf spot there may be a greyish-brown tufted fungal growth, on which spores are borne. The variable reactions of individual plants in a field suggests that there may be considerable differences in susceptibility between plants. The disease is of no economic importance in Britain and has not been studied here.

PLEOSPORA LEAF SPOT

Pleospora betae Bjorling, the fungus which causes black leg and crown rot can also infect healthy, green sugar beet leaves and produce circular, necrotic patches which are often marked with light and dark brown concentric rings (Plate XI, upper left). The spore cases (pycnidia) of the fungus are concentrated on the dark rings where they can be seen with the naked eye as minute, black dots. Usually there is no discoloration of the green leaf tissue around the lesion, which is large compared with most other leaf spots, often reaching ½ to 1 in. in diameter. This leaf spot occurs in root crops during the late summer and autumn, but does not cause appreciable damage. *P. betae* also commonly causes a brown necrosis of moribund leaves. Lesions are commonly found on the leaves of maturing seed crops. They are frequently centred upon a rust pustule on leaves of both root and seed crop plants.

SILVER LEAF

Silver leaf (caused by the seed-borne bacterium, *Corynebacterium betae* Keyw. Howell & Dowson) is a destructive disease of red-beet seed plants, particularly the variety Cheltenham Green Top; sugar beet and mangold are susceptible but infected plants have not been found in the field.

In April or May, after the plants start to bolt, occasional leaves turn silver-grey either all over or more intensely along the veins. The silvering spreads to other leaves on the stem and they eventually wilt and die. The root usually rots and the whole plant collapses. Occasional infected plants may be found in the steckling bed, and the pathogen is believed to be spread there by rain splash. It can be controlled by steeping the seed for twenty-four hours in a solution containing 200 p.p.m. of streptomycin. The disease may be confused with injury caused by wind (see p. 38)

OTHER LEAF SPOTS

A variety of necrotic lesions, sometimes resembling true leaf spot diseases, develop on the leaves of plants suffering from other diseases. They are caused by fungi* which normally infect only moribund tissues, but in atmospheric conditions favourable for infection they cause lesions on the green leaves of unhealthy plants as well as on the moribund leaves. The symptoms of these secondary infections are dealt with in the sections dealing with the primary diseases, but the following notes will act as a guide to the appropriate sections. A fern-like pattern of light and dark brown necrosis or red

* The commonest ones are *Alternaria tenuis* Auct., *Sporodesmium putrefaciens* Fuckel and *Pleospora herbarum* (Fr.) Rabenh.

or brown freckles occur on leaves infected with virus yellows. Various forms of black and brown necrotic spots occur on plants suffering from magnesium, potash and phosphate deficiencies. Yellow spots and pinkish brown angular necrotic lesions, the centres of which frequently fall out, are characteristic of leaves which have recovered from manganese deficiency.

Black lesions sometimes develop on the leaves of sugar beet in summer after they have been wet continuously for a day or two. Usually lesions are few, but sometimes fenland crops may be partly defoliated. The edges and tips of the leaves turn black and the blight progresses across the leaf, most rapidly along the veins. The plants soon recover in dry weather. The cause has not been determined, but the trouble resembles the American disease bacterial leaf spot caused by *Pseudomonas aptata* (Brown & Jamieson) Stevens. These bacteria can be seed-borne and can cause lesions on the cotyledons.

VASCULAR DISEASES

There is a group of diseases of sugar beet caused by soil-inhabiting fungi which enter through the roots and develop in the vascular tissues. Infection may discolour the vascular strands but there is rarely any rot. Symptoms usually develop on parts of the plant that are free from the fungus because it produces toxic substances which are carried through the plant and interfere with the working of the water conducting vessels. The symptoms on the overground parts are yellowing and wilting of the outer leaves which quickly die. Often one half of the leaf blade becomes yellow and necrotic whilst the other half remains green. The younger leaves are not affected at first, although all the leaves tend to have a flaccid, dull, light green appearance. In seed crops these diseases usually become apparent with the onset of hot, dry weather in summer, and cause defoliation and browning of the stems at a time when only a small proportion of the seed is fully developed. No detailed experimental work has been carried out on these diseases in this country and little is known of their occurrence.

In Holland a disease of this type has been attributed to infection with *Verticillium albo-atrum* Reinke & Berth. It first appears in July and August, when the outer leaves develop yellow patches, sometimes distributed over the greater part of the leaf. The chlorotic tissue dies quickly and the leaves droop on to the soil. The disease frequently occurs in localised areas in a crop but sometimes whole fields may be affected.

A similar disease attributed to *Fusarium conglutinans* Wollenw. var. *betae* Stewart has been reported from the United States of America. The symptoms are described as a yellowing and necrosis of the older leaves, but in addition there is a pronounced greyish-brown discoloration of one or more rings of the vascular tissues in the root.

A third disease, known to occur in Great Britain, is attributed to attacks of *Pythium* sp. on the rootlets. It is called black wood vessel disease because the characteristic symptom is a blackening of the vascular elements, so that when the root is transversely cut, concentric rings of black dots may be seen on the cut surface. The discoloration is most pronounced in the lower part of the root (Plate XIX, upper left) but it may extend as far as the crown. There is no decay of the root associated with this disease as there is with a rather similar trouble called Internal Fleck (p. 48). In the *Pythium* disease the older leaves of affected plants show a definite, interveinal chlorosis. Later

the yellow patches become necrotic; this decay involves the smaller veins, so that eventually only the main veins, with a fairly broad band of tissue on each side, are left alive. This disease has only rarely been found in the field, but roots showing black wood vessels are frequently found at beet sugar factories.

WEATHER INJURY

DROUGHT

Sugar beet plants need an ample supply of moisture throughout the season for their full development. The profuse foliage often wilts during the day in summer, and prolonged wilting may do considerable damage.

The symptoms of drought injury vary considerably. If the leaves remain flaccid for any length of time, they quickly become necrotic and bleached; the petioles remain attached to the plant and the dead leaves lie around it on the ground. When a crop is less severely affected and the leaves recover their turgidity at night, the outer ones show an indefinite yellowing, often associated with a necrosis of the tips or irregularly shaped patches on the lamina.

Occasionally the outer leaves of plants on gravelly or flinty, light soils show small, discrete, irregularly shaped, bright yellow spots; there is no necrosis and the remainder of the leaf is apparently healthy. The spots are caused by leaves wilting and touching the hot soil surface for a short period, followed by recovery with the advent of rain before serious injury has been done. The development of potash deficiency and boron deficiency is greatly accelerated by drought, and symptoms of these diseases are often found in fields in which there is severe wilting. Fusarium rot (p. 47), is another disease which occurs on growing plants only after they have been severely affected by drought.

The degree to which crops wilt varies considerably. The type of surface soil and subsoil, as well as the previous cultivations and general state of fertility, have a profound effect. In the early part of the summer, early sown crops usually wilt most readily, but later in the season late sown crops usually wilt more than those sown earlier. Crops in which there has been excessive foliage development as a consequence of heavy, unbalanced applications of nitrogenous fertilizer are particularly liable to wilt. These variations in behaviour result from differences in the rate of loss of water from the foliage by transpiration, in the water reserves in the soil, and in the extent of root development. Seed crops make particularly heavy demands on soil moisture, and dry conditions may result in premature ripening and defoliation of crops grown on light soils, particularly when the development of foliage has been forced by heavy dressings of nitrogenous fertilizer.

HAIL

Nobody seeing a sugar beet field after a heavy hail storm will be in doubt as to the nature and extent of the damage that can be caused by hail. Holes are torn in the leaf-blades; veins, midribs and leaf stalks are broken and bruised and may be practically stripped of leaf-blade. The cause of such damage may not be so obvious if leaves are seen later when they have made a partial recovery. There is considerable callus formation at the cracks, breaks

and bruises of the veins. Crops damaged by hail quickly develop fresh foliage and this recovery may be hastened by applying a top dressing of 1–2 cwt per acre of a nitrogenous fertilizer.

WIND

Strong winds tear the older foliage so that the leaves have ragged edges with a narrow band of brown, necrotic tissue bordering them. The tissue sometimes has a silvery appearance when lush, rapidly grown leaves are subjected to strong, cold winds. Frequently large leaves are folded back along the midrib in a strong wind and then the part which has the underside turned upwards is silvered. When sections of the silvered parts of leaves are examined under the microscope it is seen that a split has developed in the leaf between the epidermis and the tissues beneath, and this airspace produces the silvery appearance, in the same way as does a crack in ice.

Seed plants may be broken and blown over in heavy winds, particularly if accompanied by rain. "Ridging up" the rows of plants helps to prevent this damage. It is usually found that the plants blown over are those which have the tap roots curled up because the tails of the roots were too long when transplanted. To obviate this it is advisable to lay stecklings in rows on the ground when they are lifted and chop off the ends of the tap roots with a sharp spade.

LIGHTNING

Lightning injury occurs in more or less circular patches about five yards in diameter. The plants in the centre of the patch are most severely affected and the intensity of the damage gradually decreases from the centre outwards. This gives the impression that the crop is being attacked by a vigorous pest or disease which is spreading rapidly. This impression is accentuated by the fact that some of the outermost plants which showed no symptoms at first, begin to wilt and die later as a result of damage to the roots. The plants appear to be stunted as a consequence of the outer leaves falling to the ground, but the young heart leaves of all but the most severely affected plants remain upright and healthy. The petioles of the outer leaves are shrivelled and constricted at the base where they join the crown and consequently the leaves fall to the ground and give the impression that the plants are wilting; the outer leaves quickly die.

The symptoms on the roots vary somewhat according to their position in the affected patch and to the amount of injury inflicted (Plate XIV, upper and lower left). Some roots are black externally and furrowed with deep grooves. Almost invariably the tails of affected roots are shrivelled even though no damage is apparent elsewhere. There is usually a "point of entry" of the lightning charge near the crown or middle of the root on the side facing the centre of the affected patch of sugar beet (Plate XIV, upper right). This appears as a depressed, shrivelled area varying in extent from a small patch about half an inch in diameter to a large area embracing the whole of one side of the root right down to the tail.

Internally the roots appear in part normal, but the centres are usually permeated with cavities. The tissues in this region are whiter than usual and are streaked with black, longitudinal lines. When the internal cavities

are large there are usually thin transverse partitions of white pulverised tissue which extend across the cavity. Less severely affected roots are more or less sound in the centre with only a few longitudinal cavities. Some roots have no internal cavities, but are blackened externally and the flesh has a distinct smell of cooked beet. Such roots rapidly become slimy and decay as a result of secondary bacterial rots.

The injured plants are soon invaded by fungi, which produce a cottony, mycelial growth in the cavities in the roots. This is often a pure growth of *Pythium*, but it is not a fungus which can attack healthy beet plants of corresponding age. The most severely affected plants in the centre of the affected area die, but many of the slightly damaged ones may recover. When they do, they produce many lateral roots and develop into fangy, misshapen beets; some may be turnip-like, others bulbous above and decreasing suddenly in diameter to make a long neck which increases in diameter to give a normally shaped tail. Such roots are usually furrowed and permeated with cavities, around which there is a certain amount of decay restricted by the formation of callus tissue.

CHEMICAL INJURY

Occasionally chemicals used to control weeds in cereal and other crops get on to sugar beet, which with heavy doses are killed outright. Light doses of hormone weed killers, such as 2, 4–D, which may be blown from a nearby field or come from an unwashed sprayer used on the beet crop, cause severe distortion. Young seedlings go pale, the petioles and hypocotyl elongate and curl, and the plant becomes prostrate. In subsequent growth the petioles of several leaves fuse so that new leaves are on a tubular structure enclosing the growing point. This tube splits and later leaves are more nearly normal. Some chemicals make the leaf shape abnormal throughout the season — the lamina is frilly and instead of a single midrib, numerous more or less parallel veins run the length of the leaf. On other leaves the midrib branches to give two or three leaf-like structures on one petiole. The tap-roots may be stunted, stand out of the ground more than normal, have a rough, warty skin and an excess of fibrous roots. Unless the injury is very severe in the early stages the crop recovers.

INSECT INJURY

It is not proposed to deal in detail with any of the insect pests of sugar beet, but only to draw attention to the symptoms produced by a few, because they may be confused with the diseases described in this bulletin. Sugar beet pests are fully described in Bulletin No. 162.*

APHIDS

As well as transmitting viruses, aphids may cause direct injury to beet foliage when plants are heavily infested. Occasionally *Myzus ascalonicus* migrates from weeds on which it has lived during the winter to sugar beet seedlings, where it causes severe curling, distortion and stunting of the

* Bulletin No. 162, *Sugar Beet Pests*. Price 6*s*. 6*d*. (by post 6*s*. 11*d*.). Obtainable from H.M. tationery Office or through any bookseller.

cotyledons and young leaves. *Aphis fabae*, the black bean aphid, causes far more injury than *Myzus persicae*, the green or peach-potato aphid. Attacks of bean aphid cause leaves to crinkle, fold under, and turn yellow between the veins. Later they become necrotic at the margins and between the veins, and finally they shrivel. Usually the centre leaves are most severely affected but other leaves may also suffer. In dry, hot weather some plants are killed, but under favourable growing conditions the majority recover when the infestation ceases. The aphids leave the plants during August, but the cause of the puckered and necrotic leaves may be recognised by the cast skins of the aphids and the fungal growth on the honey dew deposited on the foliage.

CAPSID BUGS

Species of *Calocoris* and *Lygus* commonly attack sugar beet. They feed by piercing the tissues with their mouth parts and sucking the sap, thus causing the leaves and young stems of seed plants to become puckered and distorted (Plate XV, lower). Raised warts are produced on the midrib and veins of leaves where they have been punctured, and this causes a local restriction of growth which leads to distortion. The tissues inside the wart are often slightly discoloured. The tips of leaves above the warts on the main vein may become yellow, and this effect may be so pronounced that it gives the field a yellow appearance. Such heavy attacks are usually seen only in June or July, and later in the season affected crops recover. Attacks of these insects are usually more intense near thick hedges and woods from which the insects migrate in spring. The bugs, which are active, about a third of an inch in length, and bright green or brownish-green, may be found on or near affected plants. Later in the season, they may be found in considerable numbers on "bolters" in sugar beet crops, where they feed amongst the clustered, small leaves and cause considerable blistering and distortion of the young lateral stems and growing points. If the infestation is less heavy the activities of the insect are often confined to a single plant or small groups of plants.

FROG HOPPERS

The cuckoo spit insect (*Philaenus spumarius* L.) frequently attacks sugar beet, especially seed plants, during early summer. It feeds on the young leaves and exudes around itself a white foamy juice which later dries up to form a white incrustation. The leaf becomes puckered and curled around the region where the insect has fed and the adjacent leaf tissues are usually a darker and more bluish-green than normal.

MITES

In hot, dry years the foliage of sugar beet is sometimes infested with red spider mite (*Tetranychus telarius* L.). The mites feed on the underside of the leaves and are just visible to the naked eye as minute, reddish insects. The area infested may be covered with a web. The mites occur mainly on the older leaves, and the infested areas become bright yellow. The rounded patches, which are variable in size and occur indiscriminately over the leaf surface, give a pattern which is quite different from the yellowing resulting from other causes.

GENETICAL ABNORMALITIES

CHIMERAS

Occasionally sugar beet plants are found with variegated leaves. Instead of being uniformly green they have definite but irregularly shaped patches of white, yellow or greenish-white (Plate III, upper left). Sometimes the variegation looks like a white skin stretched over the green interior parts of the leaf. Occasionally plants arise which show no green tissue whatever, but they soon die. Chimeras arise as "sports"; the character which causes this phenomenon is said to be inherited in the protoplasm of the germ cells, and not on the chromosomes of the nucleus like most hereditary characters.

FASCIATION

Fasciation is the term applied to abnormalities of growth which may be described as abnormal bunching or fusing of parts of the plant. The most common type is the fusing of stems of seed plants to give wide, flat, seed-bearing stalks. Another type is the three-bladed leaf, in which three leaf blades arise from the midrib at an angle of 120 degrees from each other. There are numerous other abnormalities which are encountered from time to time. These include plants with strap-shaped leaves, excessively crinkled leaves, heart-shaped leaves, tendril-like leaves, brown scurfy, russeted roots, etc., which are not strictly examples of fasciation but may most conveniently be included here.

Root Diseases

VIOLET ROOT ROT

Light attacks of violet root rot are common on sugar beet in England and occasionally crops are seriously affected. The disease develops in crops on all types of soil, but mainly on light sandy, gravelly or peaty loams, though severe attacks sometimes occur in fields of clay loam. The disease may occasionally be found on acid soils, but it is favoured by alkalinity and is worse on calcareous soils. It is encouraged by high soil temperatures, which may be one of the reasons why it is common in the south and east of England. The distribution of infected plants in a field is often patchy. Diseased plants are sometimes concentrated in wet, badly drained hollows.

The disease is caused by a soil-inhabiting fungus, *Helicobasidium purpureum* Pat. On the surface of the root it produces a mycelial felt which is red at first, and later dark purple (Plate XVI, upper left and right). It may occur on any part of the root, but most frequently attacks the tail first and then spreads evenly up the tap root. By autumn the mycelium may have covered the root and even extended above ground, sometimes as much as an inch or two up the leaf stalks or along the soil surface, as a thick felt which is brightly coloured with zones of light purple, grey and white. If the mycelial mat is

left undisturbed until the following spring, it develops into the sporing stage of the fungus. Among the mycelium on the surface of the root there are numerous, minute, raised, round spots which are deep violet or almost black. These are infection cushions, sometimes called "corps miliaires", which consist of hard masses of mycelium each about the size of a pinhead. They develop where the mycelium penetrates the root and are a characteristic feature. Infected plants do not usually show any symptoms above ground, although in a dry autumn they sometimes wilt and appear stunted.

During the early stages of attack, and beneath the advancing edge of the mycelium, there is no obvious rotting of the root tissues. Where the mycelium is thicker and older a rot develops which is superficial at first and then more deep seated because of the action of secondary organisms. In severe attacks the roots are stunted but in most crops it does not appear greatly to affect the yield. The sugar content of diseased roots is depressed by 1–2 per cent. A further practical disadvantage of the disease is that soil clings to the roots, with the result that it increases the dirt tare. It is inadvisable to discard diseased roots in the field because infective material may in that way be distributed to parts of the field previously free from the fungus. The most satisfactory procedure in all but exceptionally bad attacks is to send both infected and healthy roots to the factory for processing. Small numbers of affected roots included in beet clamps are harmless because, although they decompose to a greater or less extent, the rot does not spread to other roots during the short time sugar beet are normally clamped in Britain. If, however, a crop containing more than about 10 per cent diseased roots is clamped, there is a danger that the clamp may heat and so suffer considerable losses. Mangolds are usually stored in clamps for much longer periods and the fungus then has the opportunity to grow from one root to another and so cause superficial infection of roots that were formerly sound.

H. purpureum produces resting bodies (sclerotia) in advanced stages of attack. They are hard, dark purple or blackish bodies which are formed on the surfaces of infected roots, particularly in the longitudinal grooves of the tap root from which most of the laterals arise. When they are formed on the finer lateral roots they are cylindrical with rounded or tapering ends, up to 1 cm in length and 3–4 mm in diameter. On the surface of larger roots, irregularly shaped sclerotium-like masses are produced which easily break up into numerous living parts when the root decomposes. In favourable conditions, sclerotia are able to survive in the soil for several years.

Many other crops besides sugar beet and mangolds are susceptible to violet root rot. The disease is frequently found in carrots, and less frequently on other arable crops such as potatoes, swedes, clover and lucerne. Many weeds which are common on arable land, pasture and leys are also susceptible. Heavy attacks of violet root rot often occur in fields which are badly infested with weeds, particularly perennials such as wild mint (*Mentha arvensis*) and yarrow (*Achillea millefolium*). The fungus not only produces resting bodies on the roots of these weeds but also persists on them throughout the winter. Severe attacks of violet root rot frequently occur in sugar beet crops taken after ploughing up old grassland. In such cases infection arises from the fungus which lived on the roots of clovers and perennial weeds in the old grass.

The principle of controlling violet root rot is to render the soil conditions as unfavourable as possible for the survival of the fungus. The conditions

favouring the survival of sclerotia are those of undisturbed moist soil, whereas cultivation, high microbiological activity and periodical drying are unfavourable. Sclerotia are produced on affected roots at a depth of at least a foot and consequently ploughing of 5–6 in. depth may leave considerable numbers unmoved. Deep ploughing and summer or bastard fallowing accompanied by frequent cultivations, dressing with farmyard and green manure are all operations which accelerate the death of the fungus. Adequate dressings of nitrogenous fertilizers also tend to reduce infection in a crop. Weed control is essential for the reduction of violet root rot because many weeds are susceptible and assist in the survival and increase of the fungus in the soil. It is bad practice to grow highly susceptible crops such as carrots and sugar beet too frequently on land which is heavily contaminated with the fungus. In some soils, however, the more thorough cultivations usually given to these crops reduces the amount of fungus and, in spite of growing susceptible crops year after year, the disease tends to die out. The fungus makes its greatest growth in late summer and autumn. Consequently, if a susceptible crop is grown which can be lifted early, such as early carrots or potatoes, it will induce sclerotia to germinate. Then, when the crop has been removed, the mycelium remaining in the soil can be starved and destroyed by bastard fallowing.

WHITE ROOT ROT

White root rot is a rare disease of sugar beet caused by *Rosellinia necatrix* Prill., a fungus more often recorded as a parasite of shrubs and trees. The fungus produces characteristic dark brown strands of mycelium in the soil and on the surface of the roots. Inside the plant the fungal strands are white.* The tissues begin to rot when they are attacked and decay increases rapidly when affected roots are clamped. The fungus produces resting bodies (sclerotia) and is said to be able to survive in the soil for considerable periods. Attacks are favoured by wet seasons, badly drained soils and high soil temperatures.

WET ROT

In summer the fungus *Phytophthora megasperma* Drechsler may attack sugar beet growing in waterlogged soil or in parts of a field which are flooded for short periods. It causes a soft rot of the root which begins at the tail and extends upwards (Plate XVI, lower right). Externally the affected parts have a greyish, shrunken appearance and internally the attacked tissues are grey or light brown. The advancing edge of the rot is dark brown and sharply marks off the diseased portion of the root from the healthy. The skin of the rotted part of the root sloughs off when the plant is pulled up and the tissues beneath are wet and slimy. Affected plants are stunted and have yellow leaves.

The disease occurs only in waterlogged soils. If the soil dries out the disease is checked and those plants which are not too severely affected may continue growth by developing fresh lateral roots above the decaying tail. This gives rise to fangy sugar beet with the remains of decay at the base of the tap root between the fangs.

* The cells of the hyphae are seen to have an unusual and characteristic pear shape under the microscope.

A similar disease which occurs in the United States is caused by *Phytophthora drechsleri* Tucker.

SCAB

Scab is common on the roots of sugar beet. The rounded, wart-like growths may be sparsely scattered over any part of the tap root or they may almost completely cover it (Plate XVII, upper). Often they are concentrated in bands around the root. Although severely affected roots are slightly stunted, growth is not much affected and scab is not considered to be economically harmful; it causes no rotting.

The disease is caused by certain soil fungi attacking the surface tissues, which react by producing a rough and corky outgrowth. The fine, light-grey threads of the fungus can sometimes be seen on the scabs on freshly lifted roots. Two types of scab have been distinguished on mangolds; raised scab, which is knob-like and results from infection with *Streptomyces tumuli* (Millard & Beeley) and pitted scab, caused by *Streptomyces scabies* (Thaxt.) Waksman & Henrici, in which the lesions tend to be depressed below the surface level of the root.

Species of *Streptomycen* are important constituents of the flora of the soil and the relative abundance of the different species varies with soil conditions. Thus, the abundance of parasitic species and the incidence of the disease caused by them depend greatly on the nature of the soil and its cultural treatment. The factors affecting the incidence of scab on sugar beet have not been investigated, but from its similarity to common scab of potato, which is also caused by *S. scabies*, it might be expected to be favoured by light, alkaline soils and restricted by acid soils, especially those with a high humus content. The disease is increased by liming and cropping too frequently with susceptible plants. It is suppressed by green manuring and by the use of acid fertilizers.

The pustular wounds made when wireworms and cutworms feed on the superficial tissues of roots often become covered with a corky layer and closely resemble scab.

GIRTH SCAB

There are numerous kinds of surface lesions on sugar beet roots the causes of which have not yet been fully investigated. Girth scab is the name given to one of these and is often found on roots grown in light, alkaline soils. At first the disease causes a superficial rough scurfiness around the middle of the root. This is composed of numerous discrete lesions which later fuse and develop into scurfy lines and areas, and these tend to run in horizontal bands around the root. In more severe attacks, the disease restricts the development of the root with the result that it develops a narrow waist, or the lower part becomes so attenuated that the root is turnip-shaped. The surface of the diseased area is depressed and has a brown, corky surface, often deeply cracked. Three species of soil fungi (*Actinomyces albus* var. *cretaceus*, *A. intermedius* and *A. nigrificans*) are reported to cause this disease, but because it is not considered to be of any economic importance in Britain, no investigations have been carried out here on its cause and control.

SCURFY ROOT

Scurfy root is the name given to a disease of adult sugar beet plants grown in acid or waterlogged soil which leads to superficial russetting and scurfiness of the tap root. Affected roots may have discrete lens-shaped surface lesions, which follow the lenticels horizontally around the root, or the surface of the whole root may be affected uniformly (Plate XVII, lower). In more severe attacks the roots may be cankered to a depth of a few millimetres and then their development is retarded. The disease is believed to be a later stage of the scurfiness of the hypocotyl which is characteristic of acid soil injury to seedlings (p. 3); the same organisms (*Fusarium culmorum*, *F. oxysporum* and others) may be isolated from the affected tissues in both diseases.

CROWN GALL

Every year a few sugar beet plants are found with a tumorous out-growth on the roots, sometimes larger than the root itself (Plate XX, upper left). These out-growths are called crown galls. They are rounded, have a furrowed, corky or warty surface, and are usually attached to the side of the root by a comparatively small neck of tissue. They are produced by the root as a reaction to infection with *Agrobacterium tumefaciens* (E.F.Sm. & Towns.) Conn. The same bacterium also infects other species of plants, including roses and fruit trees, on which galls develop at the junction between stock and scion. In sugar beet crown gall is of no economic importance, because normally only odd plants are affected, although in rare cases as many as 75 per cent of the plants in a particular field may be affected. The out-growths do not differ appreciably in sugar content from the parent roots. The tissues inside the gall are normal in colour, but they tend to rot more easily than those of a healthy sugar beet.

Another form of gall, found on sugar beet in America, is caused by infection with *Xanthomonas beticola* (Smith, Brown & Towns.) Savulescu; it is doubtful whether this bacterium is a xanthomonad. Such galls usually develop near the top of the root and are fissured and rougher than those caused by *A. tumefaciens*. In cross section they show pockets containing brown, rotted material instead of solid, white tissue as in crown gall.

Local, tumorous growths sometimes occur on sugar beet leaves (Plate XX, upper right). These have been attributed to *A. tumefaciens* but the diagnosis has not been confirmed in this country. The affected tissues become thickened and the surface rough. The edges of the growth curl inwards, but it is usually the same colour as the rest of the leaf, although it may be superficially reddened with a shiny surface.

CROWN ROT

Crown rot is a dark brown or black rot of the crown of the growing plant which, although shallow at first, may develop into a deep, vigorous rot later in the season, particularly when affected roots are clamped (Plate XVIII, upper). It is due to infection with *Pleospora betae* Bjorling, which probably gains access to the crown through the leaf scars or along a leaf stalk from a

necrotic leaf infected with the fungus. The surface of the affected part of the root is usually level with that of the healthy part, but occasionally may be slightly depressed. It may be black with a silvery sheen, or brown and corky; in the former case the surface of the decaying area is usually smooth, but in the latter it is often traversed by deep splits. The diseased tissue does not slough away as it does in the rather similar lesions of dry rot (p. 25), nor are the tissues beneath the lesion discoloured grey as in dry rot, although there is sometimes a red coloration of the tissue immediately adjacent to the rotting part. Black dots, which are the spore cases (pycnidia) of *P. betae*, can usually be found embedded in the surface of the lesion, and slimy masses of pink spores exude from them in moist conditions.

The disease is most frequently found in good crops with vigorous foliage, and is more common after wet than after dry seasons. Its importance lies not in the damage it does to the growing crop, but in the rot which develops when affected roots are clamped.

No control measures have been tested, but it is probable that a reduction of initial contamination with the fungus would prevent severe attacks. This might be attained by avoiding too frequent cropping with beet and mangolds, which result in the fungus surviving in the soil; by using seed free from infection with *P. betae* or treating infected seed with an organo-mercury compound to reduce seed-borne infection; and by placing root crops as far away as possible from seed crops in which the fungus may develop profusely in the summer, causing leaf lesions and a form of stem rot.

STEM ROT

Pleospora betae Bjorling is a common parasite of sugar beet and mangold seed crops. In addition to causing leaf spots and acting generally as an invader of damaged and moribund tissues, it causes lesions on the seed bearing stems. These lesions are usually centred on injuries such as leaf scars or the broken ends of lateral branches, and are also very common where a withered leaf clings to the stem as often happens in wet weather. The mycelium of the fungus may also travel along the leaf stalk from an infected leaf to the stem. At first, the majority of lesions are situated on the lower third of the stem, but later they tend to coalesce and then the more distal parts of the plant die and rapidly become invaded by the fungus. When the lesions remain discrete, they do not cause appreciable damage, but when attacks are severe and the lesions coalesce, the plants ripen prematurely and consequently the yield and quality of the seed is reduced. In extreme cases the whole of the base of the stem and the top of the root may be invaded by the fungus. If so, the plant withers, and the stem breaks off at the rotten crown of the root and falls over. The fungus continues to grow on the stems of the plants after harvest, especially when the crop is stacked wet. This causes infection of the seed coats which tends to be particularly heavy after wet harvests.

The lesions are at first lens-shaped, extending vertically along the stem. They have dark brown margins and grey centres, spotted with numerous minute black dots — the spore-cases (pycnidia) of the fungus (Plate XV, upper right). Larger lesions are more irregular in shape. The stems of heavily infected plants become brown, brittle and dry, and there is a prolific development of pycnidia on the parts which were first attacked. During the winter, resting-spore cases (perithecia) are produced on pieces of infected stem, and

these contain another kind of spore (the ascospore) which is discharged the following year, and which is believed to initiate infection in the following year's root and seed crops. Although the disease is always present in British crops, it is not normally sufficient to necessitate the use of special control measures, such as spraying. However, in order to minimise the risk of infection, it is advisable to burn all threshing debris on which the fungus might survive and produce its resting spore stage. In normal harvesting practice, the stems are cut off about one foot above the ground and the lower parts are left to be ploughed in. These stumps are commonly invaded by *P. betae*, either before or after harvest, and they constitute a source of future infection by means of the resting spores.

FUSARIUM ROT

Plants which have suffered severely from the effects of drought for a long period sometimes fail to recover with the onset of wetter conditions and are found to have a rot of the upper part of the root and crown (Plate XVI, lower left). This is due to infection with the fungus *Fusarium culmorum* (W. G. Sm.) Sacc., which is capable of attacking the tissues of sugar beet plants when their vitality is lowered. The fungus enters the root through the dead leaves and leaf scars on the crown. The rotted tissue is light brown, but the advancing edge of the diseased area is a darker reddish brown. When the decay has enveloped the whole of the upper part of the root, the leaves and growing points die. This may be preceded by some yellowing of the leaves around the growing point, the older leaves being prostrate and shrivelled as a consequence of the drought. The disease may be distinguished from heart rot, the advanced stages of which it closely resembles, by the fact that the growing point and young leaves are the last to succumb, whereas in heart rot they are the first. *F. culmorum* is also one of the secondary fungi which attack the crown and overground parts of plants affected with heart rot.

If more favourable growing conditions arise before fusarium rot becomes well established, the decay may be arrested and the plant recovers. The decayed tissue then becomes tough and cavities often develop in which grows a fluffy white mycelium tinged with pink and gold.

Fusarium rot develops very quickly on roots when they are clamped. Consequently if crops show signs of infection, arrangements should be made for them to be sent to the factory immediately they are lifted. Even when there is no apparent infection in the growing crop, *Fusarium* may infect the topped roots in the clamp, resulting in a disease very similar in appearance to clamp rot caused by *Botrytis cinerea*.

CLAMP ROT

Several different forms of spoilage may develop on clamped sugar beet, but that to which the name clamp rot should be applied is caused by the fungus *Botrytis cinerea* Fr. (Plate XIX, lower left). This is the most common type of rot in clamped roots in Britain. It starts either at the topped surface of the root, a broken tail or a wound on the side of the root. The disease usually develops quickly in mild weather and produces a light brown decay. When

the rot is developing vigorously, the decayed tissues merge into the healthy ones, but if conditions are not so favourable for the fungus, the advancing edge of the rot may be sharply marked off from the healthy tissues by a narrow, dark brown zone. On the surface of the decayed tissues there appears a growth of greyish fluffy mycelium which becomes darker in colour with a greenish tinge when spores are produced on it. At a later stage, resting bodies (sclerotia) develop on the surface of the root amongst the mycelium. They are first white, but later become black and hard. They measure about $\frac{1}{4}$ in. diameter and lie flattened against the surface of the root, but above they are rounded with furrowed surfaces and indented margins.

Clamp rot develops most frequently on roots which are bruised and severely injured when harvested. It also attacks roots which have been badly topped, particularly those in which the crown has been partly broken away, or which have jagged wounds caused by several cuts with the topping knife. Roots topped below the leaf scars are more liable to rot at the cut surface than those which have a few leaf buds left. The disease may spread from root to root in the clamp. The extent of the decay which develops in stored roots depends upon the length of time the roots are clamped and the temperature. If the roots have to be stored for any length of time, care should be taken to avoid excessive injury in harvesting. They should not be topped too drastically and the size of the clamp and its covering should be so arranged that the roots are kept as cool as possible without being frozen.

INTERNAL FLECK

This disease occurs in crops on fenland or low-lying fields. The exterior of affected roots may be normal in appearance or they may show a pronounced black rot extending downwards from the crown, leaving the vascular strands intact, with the softer tissue decayed. The result is that the vascular strands persist as a fibrous mat resembling the husk of a coconut. Occasionally the lesion starts lower down the root. In less advanced stages the epidermis is black and somewhat sunken. A white mycelium develops on the surface of the lesion if the affected root is kept in moist conditions.

A section of the root shows that there is a black rot of the tissues beneath the external lesion, and in other parts of the root there are flecks where the vascular tissue is affected with a dark, brown rot (Plate XIX, upper right). Small cavities are produced in the centre of many of these internal spots, often with white mycelium present. Some of the lesions appear to be quite isolated in healthy tissues, but they are all associated with the vascular bundles and so are distributed in concentric rings or arcs around the root. The lesions vary in size from minute spots to large lesions extending into the intervascular tissue in which they cause a soft black rot. When the cut surface of an infected root is exposed to the air, a pink or red coloration quickly develops around each lesion, which later changes to brown or black.

The cause of the disease needs further investigation. A species of *Pythium* has been regularly isolated from the lesions, but this fungus does not readily cause a rot in healthy sugar beet roots in infection experiments. It is believed to invade the roots through the petioles of moribund leaves touching the soil.

BACTERIAL SOFT ROT

When a well developed sugar beet is damaged above ground, as when trodden on by a horse or crushed by a tractor, the roof of the normal cavity in the crown may be broken, leaving an exposed hollow which collects rain and dew. A similar condition may also result from downy mildew infection or from insect injury to seedlings. The cavity is normally lined with a callus layer which protects the tissues from infection with parasitic organisms, but occasionally bacteria enter and cause a rot which develops throughout the root. The affected tissues are slimy and foul smelling and have a water-soaked, yellow appearance. As the rot progresses the foliage becomes yellow and later withers and dies. A similar type of rot may develop in severely damaged roots when clamped. The bacteria causing the rot have not been identified.

BLACK TAIL

Black tail is found only occasionally in Britain. The tail of the root is black, somewhat shrunken and appears to have been twisted. The blackening usually extends further on the swollen part of the root than along the groove. Internally the discoloration extends throughout the tail but higher up the root it is only superficial. The affected parts of the intervascular tissue are various tones of grey, usually a little lighter nearer the healthy tissue. The large vascular elements have a brown, woody appearance, but the smaller ones are an intense black, even beyond the area in which the tissues as a whole are discoloured. This disease was at one time attributed to infection with bacteria, but it is doubtful whether they are the primary cause of the trouble. It is probable that the bacteria invade only those roots which have been injured by flooding, drought or other agencies, that cause physiological disturbances in the plants.

FROST INJURY

The roots of sugar beet are susceptible to frost. At first affected roots have a glassy, water-soaked appearance and there are numerous splits and holes in the intervascular tissue where frost crystals have developed. The tissues may become discoloured when they thaw and the skin is often loose and easily broken. The tail is the most susceptible part. Roots most commonly affected are those laid out in rows after knocking, and then the injury extends further along the exposed side than along that resting on the ground (Plate XIX, lower right). Frosted roots decompose rapidly when they have thawed. Their appearance then depends on the organisms colonising them. Bacteria cause brown and yellow soft rots which start at the tail and extend further up the vascular tissue than the intermediate parts. The capsule-forming bacterium *Leuconostoc dextranicum* de Beijerinck also colonises frosted roots, producing a colourless, sticky slime with a characteristic vinegary, pungent odour which oozes in beads or bubbles from the surface of the root. Roots affected in this way cause serious difficulties in the beet sugar factories because their juices rapidly block the filters. The organism may also continue its activities in parts of the factory plant and cause further blockages.

Frost injury may be avoided by storing the roots in clamps during the interval between lifting and delivery to the factory. The clamps should be protected by a light covering of rough grass or hedge trimmings when exceptionally severe weather is expected. Roots which have already been exposed to frost in the field should be left in small heaps until they can be delivered to the factory. They should never be placed in large clamps because that will cause them to heat and decompose rapidly.

In severe winters a proportion of the stecklings which are out-wintered in the steckling beds or in the field after autumn transplanting, may be lost from frost injury. The largest plants are most susceptible to frost damage. If there have been appreciable losses from frost, it is often found that some survivors are also damaged and die when transplanted or make apparently normal growth at first, but suddenly wilt and die in June. Such slightly frosted plants can be recognised by the glassy, water-soaked or discoloured appearance of the internal tissue of the root, especially near the pith cavity.

LENTICEL ROT

Lenticel rot is the name given to a disease occasionally found on sugar beet roots received at the British factories, and also on clamped roots awaiting transport to the factory. The surface of the root is spotted with round or lens-shaped, black lesions about $\frac{1}{4}$ in. in diameter, which tend to be concentrated in bands around the root (Plate XVII, centre). The lesions are depressed but each one is centred on a lenticel which remains as a raised pimple. The decayed tissue is very shallow and the rot does not spread any further when the roots are kept. The cause of this trouble is not known but it closely resembles the disease of potatoes known as pit rot, which can be produced by exposing tubers to the fumes of ammonia or steeping them in the urine of rats. It seems possible that rats may be the cause of trouble in sugar beet.

INTERNAL ZONED ROT

Sugar beet roots affected with this trouble have been found only rarely and the cause of it remains unknown. Severely affected plants occur in August and can be recognised by the stunted, yellow appearance of their foliage of which only the central leaves are green and normal. Slightly affected plants show no symptoms on the overground parts and are found only by chance. Affected plants are scattered indiscriminately throughout the field. In none of the cases examined has the trouble appeared to increase after it was first discovered.

The roots of affected plants show little or no signs of external injury, though occasionally they are shrivelled or furrowed, giving one the impression that the internal tissues have shrunk or collapsed. The most outstanding feature of the disease is seen in cross sections of affected roots; these show zones of tissue coloured various tones of brown, red, yellow and green (Plate XX, lower left and right). A thin, black line usually divides the diseased, internal tissues from the healthy, external shell. Numerous cavities are present internally and, in extreme cases, the roots are hollow. Even in hollow roots, similar discoloured zones of the tissues which remain can be seen in transverse sections. In the outer tissues, and sometimes also in the

comparatively healthy tissues between the discoloured zones, there are isolated brown spots which vary in size from minute specks to zones half an inch in diameter. Microscopical examination of sections through these brown areas reveal that they consist of cells with discoloured, thickened walls, situated in the intervascular areas. The cells have split apart from one another in places, and each affected area is surrounded by a cambium layer. Isolations from affected tissues, before secondary decay has started, usually fail to show the presence of any living organisms, and when organisms have been obtained, none of them has proved to be pathogenic when inoculated into healthy roots. In many ways the symptoms of this trouble resemble those of lightning injury, but its scattered distribution in the field is quite different from the distinct circular patches caused by lightning.

EELWORM CANKER

Roots attacked by the stem and bulb eelworm, *Ditylenchus dipsaci* (Kühn) Filipjev have a slightly raised, powdery canker around the crown (Plate XVIII, lower and centre). This may be quite superficial, or it may extend throughout the whole of the upper portion of the root. The decayed tissue is dark brown and powdery or spongy. The advancing edge of the decay shows irregular lines of discoloured tissue, but there are often islets of white, healthy tissue in the decayed zone; by this feature the trouble may be distinguished from most fungal rots, which usually have a more uniform advancing edge. When examined under the microscope, the tissue from the advancing edge of the rot is found to contain many active eelworms and masses of eggs. The breakdown of the tissues is more pronounced in the intervascular areas; the vascular tissues remain white at first, but later they become discoloured and disintegrate. The initial attack may be confined to small areas around the lenticels in which case the surface of the root is covered with round or oval, pustular lesions. Rots caused by secondary fungi and bacteria often follow the eelworm attack.

The stem eelworm can invade seedlings, and either kill the growing point or distort and stunt the young leaves which often become pink. Affected plants may recover later but have swellings on the petioles and leaf veins and distorted leaves. Eventually, the roots become cankered.

Eelworm canker has been found mainly in fields lying in the valley of the River Trent, but it is also known to occur in other low-lying districts. Its distribution is patchy. The same eelworm also causes a similar decay in potato tubers, mangolds and numerous other plants. It should not be confused with the beet eelworm, *Heterodera schachtii* Schmidt; this does not cause decay of the tap root but attacks the lateral roots and produces cysts on them. Affected plants are stunted, the foliage wilts, and there is excessive growth of fibrous roots from the small tap root.

CHARCOAL ROT

Charcoal rot occurs in North America and is caused by the soil fungus *Macrophomina phaseoli* (Maubl.) Ashby, commonly known as *Rhizoctonia bataticola* (Taub.) Butl. It attacks numerous other crops, such as maize, beans and sweet potatoes. It is an active parasite only at high temperatures and consequently it is limited to those districts in North America where extremely high summer temperatures are experienced. The fungus is a wound parasite

and charcoal rot is rarely very serious. It begins as a brownish-black rot in wounds around the crown, but as the rot advances the root is reduced to at dry mass resembling charcoal, covered by a thin, dry, papery shell.

DRY ROT CANKER

The fungus *Corticium solani* (Prill. & Delacr.) Bourd. & Galz., which infects seedlings and young beet plants, also causes a rot of fully grown sugar beet in North America. Infection usually starts at cracks or around the bases of lateral roots. Externally, affected roots have a woody appearance and are marked with fine concentric rings. Beneath the dry, sunken lesions on the surface there are deep pockets of brown, spongy, decayed tissue, sharply delimited from the healthy parts of the root. Open cankers may develop and, as the beet enlarges, these become large open cracks. Control measures consist of reducing soil contamination by suitable crop rotations, avoiding sugar beet, beans, potatoes and other susceptible crops too often in the rotation.

SOUTHERN SCLEROTIUM ROT

Southern sclerotium rot is a serious disease of sugar beet in North America which is favoured by moist, warm soil conditions. It is caused by the fungus *Sclerotium rolfsii* Sacc., the white, cottony mycelium of which covers the surface of affected roots. Among the mycelium, resting bodies (sclerotia) about the size and colour of mustard seeds are produced. They remain viable in the soil for several years and are spread from field to field in soil carried on implements, vehicles and plants. Special precautions are necessary for dealing with loads of beet from fields contaminated with the fungus. The disease spreads rapidly in an infected crop.

Apart from the precautions taken to avoid contaminating land with the resting bodies of the fungus, control methods involve the growing of winter crops (which is possible in the climate where the disease occurs) and fallowing the land in the summer, in order to reduce the numbers of sclerotia in the soil to a safe level for growing sugar beet. Other susceptible crops, such as carrots, beans and potatoes, are avoided. Nitrogenous fertilizers provide a partial control in many fields. Soil samples from fields in which the disease has occurred are examined before further susceptible crops are taken, in order to determine whether the number of living sclerotia has been reduced to a safe level.

The disease also occurs in southern Europe and the Azores, where the causative fungus has been wrongly described as various species of *Typhula*.

RHIZOPUS ROOT ROT

Rhizopus root rot is a disease of growing beets caused by *Rhizopus arrhizus* Fischer which has been reported from North America. Affected plants wilt without changing the colour of the leaves, and in the roots there is a greyish-brown, spongy decay smelling of vinegar, which usually begins near the tail and extends upwards. Soil clings to affected roots and a mantle of white fungus mycelium develops around the shoulder. The distal parts of the lateral roots may remain unaffected until they are cut off by decay from the main root. The disease is favoured by high temperatures. *R. oryzae* Went & Pr. Geerlings also behaves as a wound parasite at temperatures of 30–35°C and *R. nigricans* Ehrb. at 14–16°C.

Index

(Page numbers of main references are shown in heavy type)

	Page
Achillea millefolium	42
Acid soil (see Soil acidity)	3, 7, 20, 25, 28, 41, 44
Acid soil injury	3, 6, 44, 45
Actinomyces albus var. *cretaceus*	44
Actinomyces intermedius	44
Actinomyces nigrificans	44
Agallia albidula Uhl.	19
Agalliana ensigera	19
Agrobacterium tumefaciens	45
Alkaline soil (see Soil alkalinity)	24, 27, 28, 41, 44
Alternaria	23
Alternaria tenuis	11, 35
Aphanomyces cochlioides	1
Aphis fabae	8, **13**, 40
Argentine Curly Top	**19**
Atanus exitiosus	20
Bacterial Blight	**36**
Bacterial rots	8
Bacterial Soft Rot	**49**
Basic slag	25
Beans	51
Beet Ringspot	**19**
Beta maritima	12, 31
Black bean aphid	13, 14
Black Leg	1, 6, 21, 35
Black Tail	**49**
Black Wood Vessel Disease	**36**
Blind Seedlings	**6**
Borax	28
Bordeaux mixture	31, 34
Boron deficiency (see also Heart Rot)	7, 8, **25**, 37
Botrytis cinerea	47
Brassicae (-a crops)	12, 13
Brazilian Curly Top	**19**
Bryonia dioica	18
Calcium deficiency	10, **28**
Caloxoris sp.	40
Capsid Bug injury	7, 9, **40**
Carrots	42, 52
Cercospora beticola	**34**
Cercospora Leaf Spot	9, **34**
Charcoal Rot	**51**
Chemical Injury	39
Chenopodium album	12, 17, 31
Chicken and Hen	1
Chickweed	12
Chimeras	7, 10, **41**
Chrysanthemum segetum	3
Clamps (see also Mangold clamps)	27, 42, 43, 47, 49, 50
Clamp Rot	**47**
Clover	42
Copper deficiency	**28**
Corn Mangold	3
Corticium solani	1, 52
Cover crops	15
Crinkle	**20**
Crop rotation	34
Crown Gall	**45**
Crown Rot	35, **45**
Cucumber mosaic	**18**
Curly Top	**19**
Cutworms	44
Downy Mildew	7, 8, **29**, 49
Symptoms	29
Effect on yield	30
In seed crops	30
Life Cycle	30
Control	31
Drab Disease	21
Drought injury	8, 9, 37
Dry Rot (see also Heart Rot)	8, **25**, 46
Dry Rot Canker	**52**
Dwarf	9, **18**
Eelworm, Beet	51
Eelworm Canker	**51**
Erysiphe polygoni	32
Euonymous europaeus	13
Eutettix tenellus	19
Fangy roots	3, 43
Farmyard manure	22, 23, 25, 27, 43
Fasciation	**41**
Fluoroacetamide	16
Froghoppers	**37**
Frost injury to seedlings	5, **32**
Frost injury to roots	49
Fusarium conglutinans var. *betae*	36
Fusarium culmorum	3, 45
Fusarium oxysporum	3, 45
Fusarium Rot	37, **47**
Fusarium sp.	26
Gamma-BHC	2, 3
Garden beet (see also Red beet)	12, 17, 31
Girth Scab	**44**
Goosefoot	12, 17, 31
Grey Speck in oats	24
Groundkeeping beets	15, 18, 31, 33
Groundsel	12

	Page
Hail injury	**37**
Heart Rot (see also Boron deficiency and Dry Rot)	8, **25**, 28, 29, 47
Helicobasidium purpureum	41
Heterodera schachtii	51
Hover flies	13
Hyperomyzus staphyleae	15
Insect injury	6, 7, 8, **39**
Internal Fleck	36, **48**
Internal Zoned Rot	**50**
Iron deficiency	7, 10, **28**
Ladybirds	13
Leaf Curl	**20**
Leaf Spots	8, 9, 11, **33**
Lenticel Rot	**50**
Leuconostoc dextranicum	49
Lightning injury	7, 8, **38**, 51
Lime, liming	4, 23, 24, 27, 28
Lucerne	42
Lygus sp.	40
***M**acrophomina phaseoli*	51
Magnesian limestone	24
Magnesium deficiency	7, 8, 10, **23**, 36
Magnesium sulphate	23
Maize	51
Manganese deficiency	7, 9, **24**, 36
Manganese sulphate	24
Manganese toxicity	3, 7, 10, **25**
Mangolds	2, 15, 18, 31, 42, 44, 46, 51
Mangold clamps	12, 15, 18, 33
Marrow	18
Marsh Spot in peas	24
Mentha arvensis	42
Microsphaera betae	32
Mites	8, 9, **40**
Mosaic	7, 9, **17**, 18, 28
Muriate of potash	22
Myzus ascalonicus	39
Myzus persicae	**12**, 18, 40
Nitrate of soda	4, 22, 27
Nitrogen deficiency and excess	7, 8, 10, **20**
Nitrogen (-ous fertilizer)	4, 16, 20, 27, 33, 37, 43
Oats	24
Organo-mercury fungicide	2, 34, 46
Pea	24
Peach	13
Peach-Potato aphid	**12**, 18, 40
Peronospora schachtii	29
Philaenus spumarius	40
Phoma betae	2
Phosphate deficiency	3, 10, **21**, 36
Phosphatic fertilizer (superphosphate)	3, 21, 27
Phytophthora drechsleri	44
Phytophthora megasperma	43
Piesma cinerea	20
Piesma quadrata	20
Pit Rot of potatoes	50
Pitted Scab	44
Plant population	6, 16
Pleospora betae	2, 26, 35, 46, 47
Pleospora herbarum	35
Pleospora Leaf Spot	10, **35**
Potash deficiency	7, 9, 10, **21**, 36, 37
Potash fertilizer	22
Potatoes	43, 44, 50, 51, 52
Powdery Mildew	8, 9, **32**
Pseudomonas aptata	36
Purity of root juices	12, 30, 34
Pythium sp.	36, 48
Pythium intermedium	1
Pythium ultimum	1
Pythium Yellows	8, **36**
Raised Scab	**44**
Ramularia beticola	34
Ramularia Leaf Spot	9, 34
Rats	51
Red beet (see also Garden beet)	12, 17, 31
Red spider	8, 40
Resistant varieties	16, **19**, **31**, **33**, 34, 35
Rhizoctonia bataticola	**51**
Rhizoctonia solani	1
Rhizopus arrhizus	52
Rhizopus nigricans	52
Rhizopus oryzae	52
Rhizopus Root Rot	**52**
Rosellinia necatrix	43
Rumex acetosella	3
Rust	8, 9, 11, **33**
Salt	22, 23, 27, 28
Savoy cabbage	12
Savoy disease	20
Scab	**44**
Sclerotium rolfsii	52
Scorch	21
Scurfy Root	45
Seakale beet	12, 31
Seed treatment (see Organo-mercury fungicide)	2, 34, 46
Sheep's sorrel	3
Silver Leaf	**35**, 38
Snowball tree	13
Sodium deficiency	22
Soil acidity	**3**, 7, 20, 25, 28, 41, 44, 45
Soil alkalinity	4, 24, 25, 28, 41, 44
Soil erosion	5
Southern Sclerotium Rot	**52**
Speckled Yellows	**24**, 28
Spergula arvensis	3
Spinach	12, 17
Spindle bush	13
Sporodesmium putrefaciens	11, 35
Spurrey	**3**

	Page
Stellaria media	12
Stem Eelworm	5, 51
Stem Rot	**46**
Strangles	6, 7, 8
Streptomyces	44
Sugar content	12, 19, 21, 25, 27, 30, 34, 42
Sulphate of ammonia	22, 25, 27
Sulphur	25
Swede	42
Sweet potato	51
Systematic insecticide	16, 18
Tetranychus telarius	40
Tomato	18
Typhula sp.	52
Uromyces betae	33
Variegation	41
Vascular diseases	7, **36**
Verticillium albo-atrum	36
Viburnum opulus sterile	13
Violet Root Rot	**41**
Virus Yellows	7, **8**, 17, 18, 21, 23, 29, 32, 33, 36
Virus Yellows—*continued*	
Symptoms	8
Effect on yield	11
Sources of infection	12
Vectors	12
Control	13
Wasps	13
Weeds	3, 12, 17, 18, 19, 20, 25, 34, 42
Weed killers	16, 39
Wet Rot	8, **43**
White bryony	18
White Root Rot	**43**
Wild beet	12, 31, 33
Wild mint	42
Wind erosion	5
Wind injury	5, 6, 38
Wireworms	3, 44
Yarrow	42
Yellow Net	**17**
Yellow Wilt	**19**
41-yellows	**17**
Zinc deficiency	28

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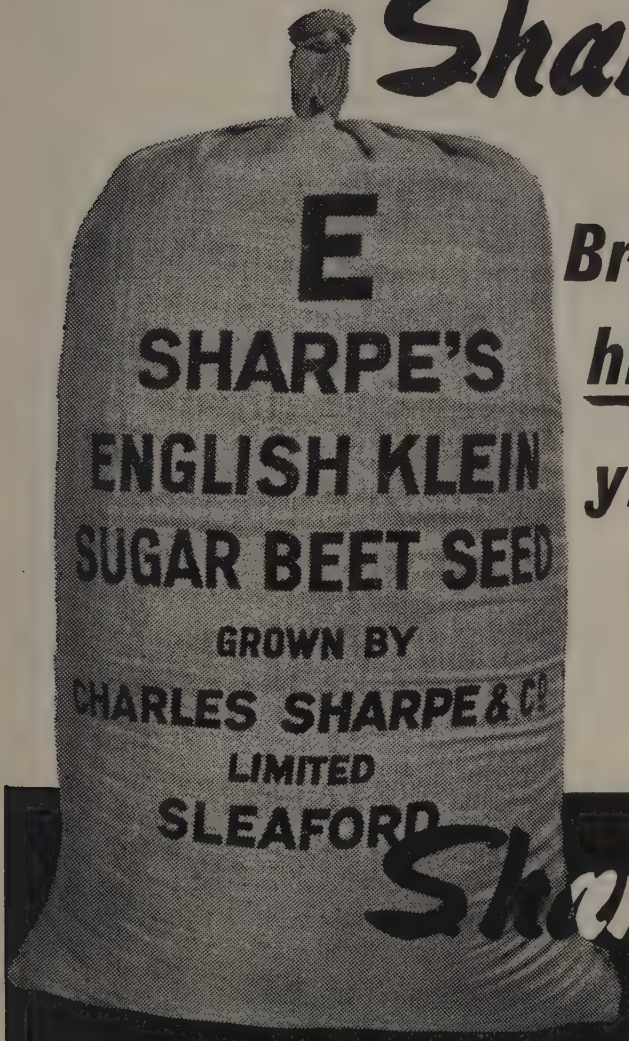

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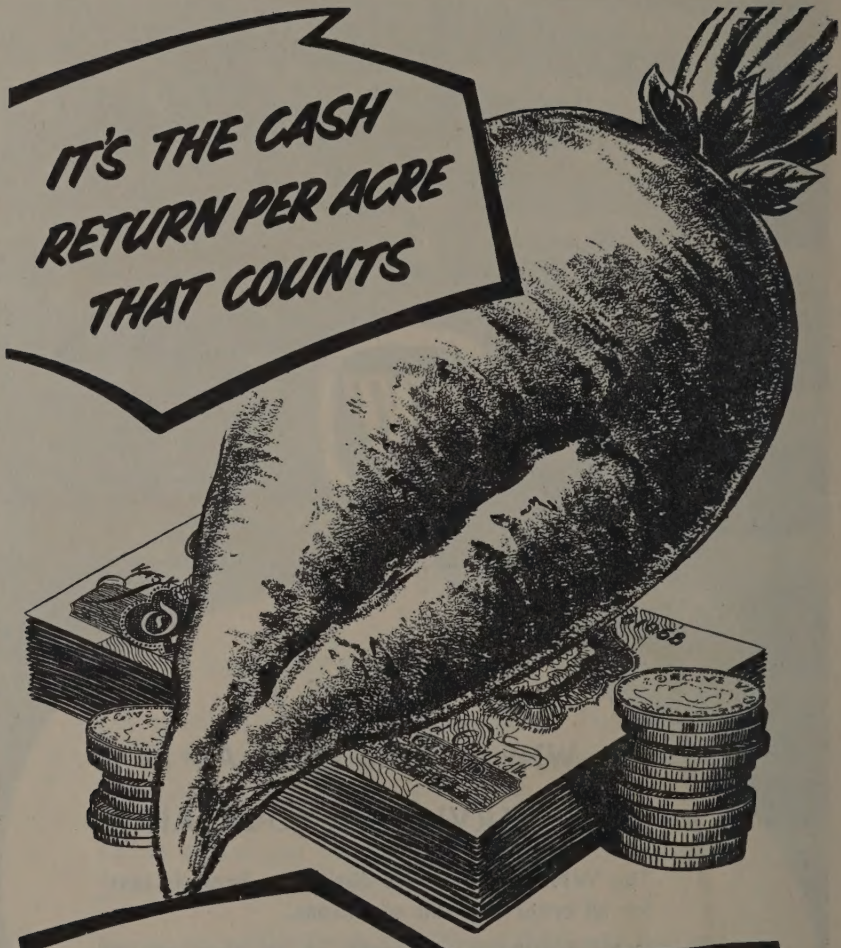

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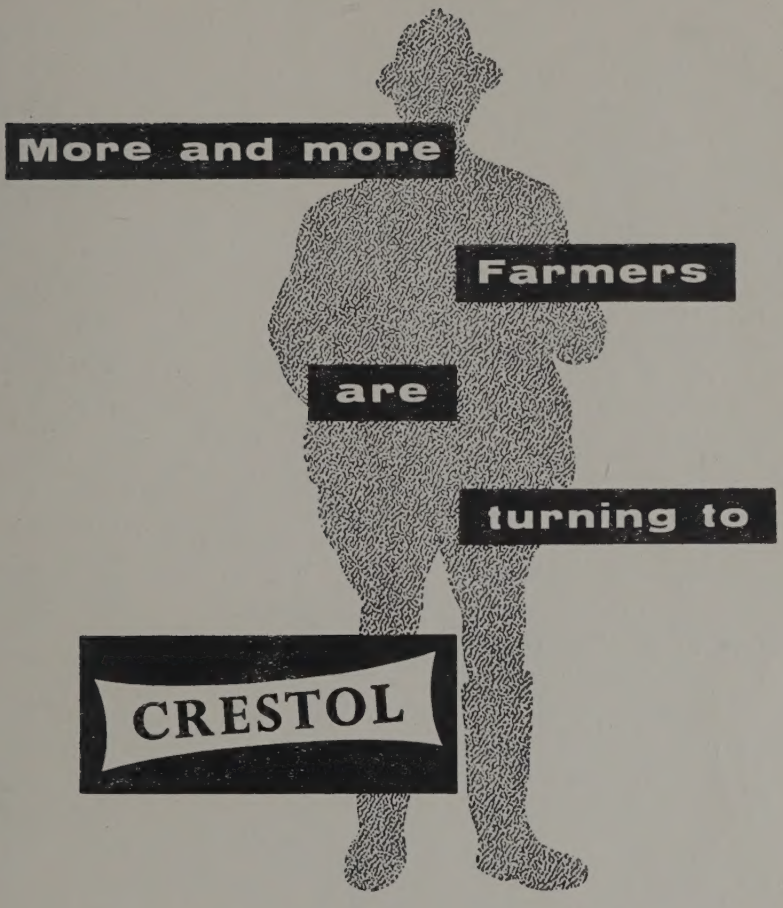

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